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FIG. I.

Fig. I. Arteriosclerosis of the thoracic and abdomina, aorta, showing irregular nodules, atheromatous plaquesl denudation of the intima, thin plates of bone scattered throughout with spicules extending into the lumen of the vessel. Note the contraction of the openings of the large branches, the rough appearance of the aorta and the greater degree of sclerosis of the upper two-thirds, i. e., of the aorta above the diaphragm. This aorta in the recent state was much thickened and almost inelastic.

Arteriosclerosis:

Etiology, Pathology, Diagnosis, Prognosis, Prophylaxis, and Treatment.

By

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With an introduction

by

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Eight original illustrations.

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St. Louis

This volume is affectionately dedicated to my mother.

—The Author.

PREFACE.

It is hoped that this small volume may fill a want in the already crowded field of medical monographs. Our attempt has been to give to the general practitioner a readable, authoritative essay on a disease which is especially an outcome of modern civilization. To that end all the available literature has been freely consulted, and the newest results of experimental research and the recent ideas of leading clinicians have been summarized. The writer has supplemented these with results from his own experience but has thought it best not to burden the contents with case histories.

The stress and strain of our daily life has, as one of its consequences, early arterial degeneration. There can be no doubt that arterial disease in the comparatively young is more frequent than it was twenty-five years ago, and that the mortality from diseases directly dependent on arteriosclerotic changes is increasing. Fortunately, the al-

most universal habit of getting out-of-doors whenever possible, and the revival of interest in athletics for persons of all ages, have to some extent counteracted the tendency to early decay. Nevertheless, the actual average prolongation of life is more probably due to the very great reduction in infant mortality and in deaths from infectious and communicable diseases.

The wear and tear on the human organism in our modern way of living is excessive. Hard work, worry, and high living all predispose to degenerative changes in the arteries and so bring on premature old age.

We have tried to emphasize this by laying stress on the prevention of arteriosclerosis rather than on the treatment of the fully developed disease.

No bibliography is given inasmuch as this is not intended as a reference book but rather as a guide to a better appreciation and understanding of a most important subject. It has been difficult to keep from wandering off into full discussions of conditions incident to and accompanied by arteriosclerosis; but, in order to be clear in our statements and complete in our descriptions, we have had

to invade the fields of heart disease, kidney disease, brain disease, etc. We trust, however, that these excursions will serve to show how intimately disease of the arteries is bound up with diseases of all the organs and tissues of the body.

Some authors have been named when their opinions have been given. Thanks are extended also to many others to whom the writer is indebted but of whom no individual mention has been made.

The writer also takes this opportunity of expressing his appreciation of the kindness of Dr. D. L. Harris, who took the microphotographs, and to the publishers for their unfailing courtesy and consideration.

L. M. WARFIELD.

St. Louis, Mo., August, 1908.

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INTRODUCTION.

There is a despotism to which the greater part of mankind is enslaved, a despotism as absolute in the republic as in the autocracy —the tyranny of words. The thought or fancy, unexpressed, may have its passing influence; expressed, the mere sound of our own voice exercises upon us a subtile influence which, as it were, drives home the idea, while repetition fastens upon us an impression which, before we are aware of it, has become a conviction—a part of ourselves.

A term which strikes the popular ear becomes soon associated, in the mind of the average individual, with an idea or a picture which may vary greatly from that of his neighbor, and more yet from the truth. Nevertheless time and repetition fix the idea until 'tis difficult to realize that the word has not to everyone the same constant and sharply defined signification.

The prevalence of such popular words and expressions in medicine is familiar to all.

These vary greatly in character and origin. Those of older years were usually expressions intended to describe certain groups of clinical symptoms and were based largely on purely hypothetical considerations. These terms must, of necessity, have been rather indefinite and uncertain in their application even among the medical profession, and much more so among the general public. Such is that commonest and most detestable of words which means everything to everybody and nothing under the sun in itself, "Biliousness." Such has been the term "Malaria" in its popular sense. Such is, often enough, the all too popular word "Rheumatism." In more modern times, with the development of knowledge of pathological anatomy and physiology, more accurate terms have come into medicine, some based on anatomical, some on physiological changes. Many of these terms have also passed over into popular usage. And while, originally, they designated specific anatomical conditions or physiological processes, the uninstructed public associate them naturally with groups of symptoms, and form many and varied

ideas as to their significance. But to each individual the words mean something.

All this has too often its repercussion on the physician who, in order to satisfy his patients, who demand a name for the symptoms from which they suffer, is led, almost unconsciously, to use a specific term in a general way to cover a variety of conditions in which perhaps the exact diagnosis may not be wholly clear, until, by force of habit and repetition, he finds a certain satisfaction in hiding behind an empty term, and becomes himself a victim of the tyranny of words. What an array of pathological processes have been dismissed under the specific diagnosis of "gastritis" or "neuritis"!

The study of those changes in the blood vessels, hyperplastic, degenerative, or inflammatory which are the inheritance of advancing years, and have been so aptly called "the rust of life", is not new. The term "Arteriosclerosis" was used anatomically by Lobstein three quarters of a century ago, and the relations of arterial change to visceral disease have long been a fertile field for speculation and study. But the popularization of the term "Arteriosclerosis" from a clinical standpoint

is relatively recent. In later years, however, it has definitely caught the popular ear; it figures in the newspapers as a "new disease;" it means something to each member of the public; it is a diagnosis satisfying to the anxious friends of the patient. And, too often, the general diagnosis "Arteriosclerosis" has come to satisfy the physician himself who, without finding a definite explanation of the obscure symptoms of his patient, rests on his oars with the constatation of the tortuous temporal or the palpable radial of the sufferer. The term "Arteriosclerosis" is fast coming to take a place near the throne once occupied by "Malaria"; it is becoming a dangerous word.

Great as is the importance of arterial changes in relation to many of the ills to which flesh is heir, and numerous as have been the anatomical, clinical and experimental researches concerning this subject, it must be acknowledged that there is much yet to be learned with regard to the etiology, the manner of development, the nature of the changes in different parts of the arterial tree, their relation to variations in blood pressure and to visceral disease as well as concerning the relations of peripheral to central changes;

and there are still wide differences of opinion as to the interpretation of some of the observations which have been made.

In view therefore of these considerations as well as of the widespread and indiscriminate popular use of the term "Arteriosclerosis," the time would seem to be peculiarly fitting for the publication of a brief and practical consideration of the present state of our knowledge concerning the nature and clinical bearings of arterial disease such as that which my friend Dr. Warfield seeks to set forth.

There can be no doubt that we Americans are prone to waste our energies. We do not know how to rest or to conserve our strength; and it is probably true, as Dr. Warfield suggests, that the wear and tear of this feverish and unreasoning activity leaves, too often, an early mark on the cardio-vascular system.

It should, as he has said, be the earnest endeavor of the physician to prevent the premature development of these vital changes rather than to seek to alleviate symptoms after irreparable damage has been done.

William Sydney Thayer.

Baddeck, Cape Breton, 26 August, 1908.

CHAPTER I.

ANATOMY.

With the increased complexity of our modern life, comes increased wear and tear on the human organism. This is most often revealed in the very commonly seen arterial disease which may develop in persons much under middle age. The old adage that "A man is as old as his arteries" is even more true today, if possible, than when it was first said.

Arteriosclerosis is not universally considered to be a disease *sui generis;* some authors would rather call it a symptom or a group of symptoms which, however, are not specific enough to warrant their collection into a definite disease. We shall, however, consider arteriosclerosis as a disease rather than as a symptom group for, from a clinical standpoint, there is much in favor of regarding it as an entity.

Definition—Arteriosclerosis (Arteriocapillary fibrosis) may be defined as a subacute and chronic disease of the arteries, char-

acterized anatomically by increased thickness of the walls of the blood vessels, the initial lesion being for the most part in the middle (muscular) coat, leading not infrequently to calcification of this coat and to the formation of minute aneurysms along the vessels. The term arterio-capillary fibrosis undoubtedly has a broader meaning. Almost without exception the capillaries are involved in the morbid process, and even the veins may be markedly thickened. Under such circumstances, it is proper to speak of vascular sclerosis or angiosclerosis.

A few brief reminders of the anatomy of the arteries will not be out of place here. For the clear comprehension of the disease under discussion, it is necessary to keep in mind the essential histological differences between the aorta and the larger and smaller branches of the arterial tree.

The vascular system, as a whole, is often referred to as a central pump with a series of closed tubes that branch widely, and, collecting again, converge into vessels of the same area as at the beginning. While this is a rough illustration, it is useful but natur-

FIG. II.

Fig. II. Normal aorta. Compare with Figure I. Note the perfectly smooth, glossy appearance of the intima. The openings of all the intercostal arteries are distinctly seen. In the recent state this artery was highly elastic, capable of much stretching both transversely and longitudinally.

ally does not take into account the vital forces that control every part of the system.

General Structure of The Arteries — The essential portion of any blood vessel is the endothelial tube composed of flat cells cemented together by intercellular substance and having no stomata between the cells. This tube is reinforced in different ways by connective tissue, smooth muscle fibres and fibro-elastic tissue. Although the gradations from the larger to the smaller arteries and from these to the capillaries and veins are almost insensible, yet particular arteries present structural characters sufficiently marked to admit of histological differentiation.

The whole vascular system, including the heart, has an endothelial lining which may constitute a distinct inner coat, the tunica intima, or may be without coverings as in the case of the capillaries. The intima (Fig. III.) consists typically of endothelium reinforced by a variable amount of fibro-elastic tissue in which the elastic fibres predominate. The tunica media is composed of intermingled bundles of elastic tissue, smooth muscle fibres, and some fibrous tis-

sue. The adventitia or outer coat is exceedingly tough. It is usually thinner than the media and is composed of fibro-elastic tissue. This division into three coats is, however, somewhat arbitrary, as in the larger arteries, particularly, it is difficult to discover any distinct separation into layers.

The muscular layer varies from single scattered cells, in the arterioles, to bands of fibres making up the body of the vessel in the medium sized arteries and veins.

There is elastic tissue in all but the smallest arteries, and it is also found in some veins. It varies in amount from a loose network to dense membranes. In the intima of the larger arteries the elastic tissue occurs as sheets which under the microscope appear perforated and pitted, the so-called fenestrated membrane of Henle.

The nutrient vessels of the arteries and veins, the vasa vasorum, are present in all the vessels except those less than one millimeter in diameter. The vasa vasorum course in the external coat and send capillaries into the media. Lymphatics and nerves are also found in the vascular walls.

ARTERIES—The structure of the arteries varies notably, depending upon the size of the vessel. In a cross section of the radial artery, one sees a wavy outline of intima, caused by the endothelium following the corrugations of the elastica. The endothelium is seen as a delicate line in which a few nuclei are visible. The media is comparatively thick, and is composed of muscle cells, arranged in flat bundles, and plates of elastic tissue. Between the media and the externa the elastic tissue is somewhat condensed to form the external elastic membrane. The adventitia varies much in thickness, being better developed in the medium sized than in the large arteries. It is composed of fibrous tissue mixed with elastic fibres.

"Followed towards the capillaries, the coats of the artery gradually diminish in thickness, the endothelium resting directly upon the internal elastic membrane so long as the latter persists, and afterwards on the rapidly attenuating media. The elastica becomes progressively reduced until it entirely disappears from the middle coat, which then becomes a purely muscular tunic and, before

the capillary is reached, is reduced to a single layer of muscle cells. In the precapillary arterioles the muscle no longer forms a continuous layer, but is represented by groups of fibre-cells that partially wrap around the vessel, and at last are replaced by isolated elements. After the disappearance of the muscle-cells, the bloodvessel has become a true capillary. The adventitia shares in the general reduction, and gradually diminishes in thickness until, in the smallest arteries, it consists of only a few fibro-elastic strands outside the muscle-cells" (Piersol's Anatomy).

The large arteries differ from those of medium size mainly in the fact that there is no sharp line of demarcation between the intima and the media. There is also much more elastic tissue, distributed in firm bundles throughout the media, and there are fewer muscle fibres, giving a more compact appearance to the artery as seen in cross section. This predominance of elastic tissue permits of great distension by the blood forced into the artery at every heartbeat, the calibre of the tube being less markedly under the control of the vasomotor nerves than is the case

FIG. III.

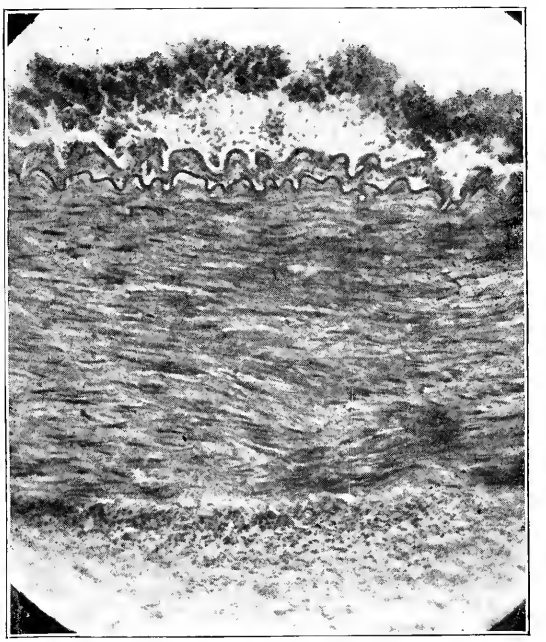

Fig. III. Cross section of a large artery showing the division into the three coats; intima, media, adventitia. The intima is a thin line composed of endothelial cells. The wavy elastic lamina is well seen. The thick middle coat is composed of muscle fibres and fibro-elastic tissue. The loose tissue on the outer (lower portion of cut) side of the media is the adventitia.

in the small arteries where the muscle tissue is relatively more developed. The adventitia of the large arteries is strong and firm, and is made up of interlacing fibro-elastic tissue of which some of the bundles are arranged longitudinally.

VEINS—The walls of the veins are thinner than those of the arteries; they contain much less elastic and muscular tissue, and are, therefore, more flaccid and less contractile. Many veins, particularly those of the extremities, are provided with cuplike valves opening towards the heart. These valves, when closed, prevent the return of the blood to the periphery and distribute the static pressure of the blood column. The bulgings caused by the valves may be seen in the superficial veins of the arm and leg. There are no valves in the veins of the neck where there is no necessity for such a protective mechanism, gravity sufficing to drain the venous blood from the cranial cavity.

CAPILLARIES—These are endothelial tubes in the substance of the organs, the tissue of the organ giving them the necessary support. They are the final subdivisions of the blood-

vessels, and the vast capillary area offers the greatest amount of resistance to the blood flow, and thus serves to slow the blood stream and allow time for nutritive substances or waste products to pass from and to the blood. Usually the capillaries are arranged in the form of a net work, the channels in any one tissue being of nearly uniform size, and the closeness of the mesh depending upon the organ. Thus, in the lung, the mesh work is closest; in organs of great functional activity, as in the kidneys, the thyroid, the liver, etc., there is an enormous capillary net work.

The capillaries have no nerve supply, but are flushed or emptied entirely by the dilatation or contraction of the small arteries. The capillary resistance really depends to a great extent on the behaviour of the very small arteries in which are a few muscle cells sufficient to close the lumen of the vessel when excessively stimulated and thus to shut off a capillary area. When this happens to great numbers of the smallest arteries, parts of organs or whole organs may be rendered anemic, and, in the case of the fingers or toes, small portions may actually become gangrenous.

CHAPTER II.

PHYSIOLOGY OF THE CIRCULATION.

BLOOD PRESSURE.

No attempt will be made to give more than a very brief outline of the chief points in the physiology of the circulation that have a special bearing on the disease under discussion. A complete understanding of the physiology of the heart and bloodvessels facilitates the comprehension of many of the morbid processes that are found in arteriosclerosis and explains why it is that not always do we have defects that *a priori* might be expected to follow certain causes.

"The heart and the blood vessels form a closed vascular system containing a certain amount of blood. This blood is kept in endless circulation mainly by the force of the muscular contraction of the heart. But the bed through which it flows varies greatly in width at different parts of the circuit, and the resistance offered to the moving blood

is very much greater in the capillaries than in the large vessels." (Howell, Textbook on Physiology.)

The velocity varies greatly in different parts of the circulation. In the arteries it changes with every heart beat. As the bed widens the flow is necessarily slower, until, in the enormous capillary area, the flow has become even and slow, and increases as the blood is collected into the venules, until at the venous openings of the right auricle it is almost as rapid as at the aorta.

Innumerable factors influence the rate and amount of the flow to any part or parts. Given a fixed amount of fluid that completes a circle in a certain time, any change in any part of the circle necessarily has its opposite effect elsewhere in the circle provided we assume that the force and rate of the heart beat remain the same. Thus contraction of all the vessels in the splanchnic area cuts off an enormous portion of the circle. Now, if the blood is to make the circuit in the same time as before the contraction, then vessels elsewhere must dilate to a corresponding degree. Suppose now that there were some

substance in the circulation that acted particularly on the musculature of the splanchnic vessels causing them to contract and to a lesser extent on the other arteries, then an increased resistance would result which could be overcome only by more force exerted on the part of the heart.

At every systole from 50 to 100 cc. of blood are thrown into an already filled aorta. There is thus 70–80 times a minute forcible stretching of the arch and the thoracic aorta. In order to accommodate this extra blood these structures must expand. After the closure of the aortic valves the column of blood is kept at a considerable pressure by the compression of the highly elastic aorta. At the top of the arch three large vessels are given off, the innominate, the left carotid, and the left subclavian. These to some extent take up a portion of the strain. No other large vessels are given off until below the diaphragm. It is thus seen why it is that the thoracic aorta is more apt to be the seat of disease than any part of the vascular system.

In order that the blood may course through the vascular system a certain head of pres-

sure must be maintained. As the heart contracts, the pressure is suddenly raised and the pulse wave is transmitted towards the periphery. This pressure is known as the maximum or systolic pressure. It is, of course, highest at the heart. In the brachial artery in man, where the pressure is usually measured, it amounts to 100 to 130 mm. of mercury. It is lower in children and higher in old people as a general rule, to which there are numerous exceptions. The minimum pressure in the artery, i. e., the pressure at the end of diastole, is known as the diastolic pressure. The difference between these two pressures is known as the pulse pressure, and measures normally in an adult in the brachial artery about 45 to 48 mm. of mercury. This represents to a great extent the elastic force of the aorta which during diastole keeps the blood flowing and maintains the pressure.

Several instruments have been devised to measure the maximum blood pressure in man. Instruments have also been made to measure the minimum pressure but they are too complicated for general use. Cook's modification of the Riva-Rocci instrument with the 9 or

FIG. IV.

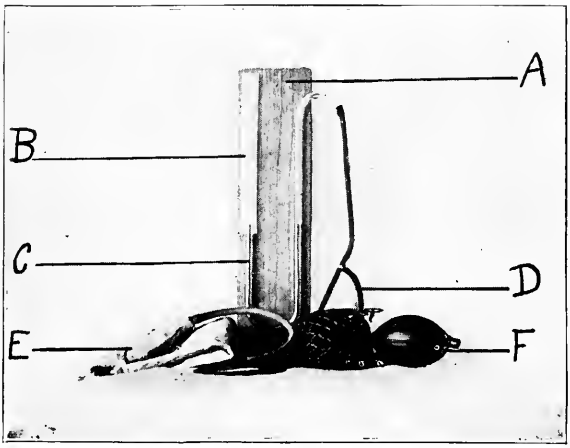

Fig. IV. A convenient sphygmomanometer. A, stand made of thin board fitted securely to a heavy base 4x4x¾ in.; B, scale graduated in centimeters and millimeters from 1 to 15; C, the U-tube of heavy glass 2 mm. in diameter, the mercury shown standing at zero on the scale; D, short outlet tube with clamp; E, 9 cm. arm band of rubber encased in heavy canvass, the hooks and eyes sewn on the canvass so that the band may be snugly fitted around the arm above the elbow; F, a double bulb syringe for inflating the arm band. The arm band and tube containing the mercury have free air connection so that when the arm is compressed by the air forced into the arm band the mercury rises in the tube. With a finger on the pulse of the subject the syringe is compressed until the pulse disappears. By cautiously letting out air through the clamp D, the point on the scale is noted when the pulse first is felt. Several readings are to be made and the average taken. This gives the systolic pressure. The diastolic pressure cannot be determined with any degree of accuracy with any but the most elaborate instruments. The distance that the mercury rises in the left arm of the tube is doubled in order to get the whole reading, for the mercury in the tube on the right is depressed to the same extent as it is elevated on the left. This instrument enables one to read pressures of 300 mm. of Hg.

12 cm. arm band, Janeway's portable instrument or the Stanton instrument are all used. The principle upon which all depend is the compression of the brachial artery by means of air forced into a band, resulting in the obliteration of the pulse at the wrist. Mercury is forced into an upright, or U-tube, and just at the time when the pulse at the wrist disappears, the reading is made. This is the systolic pressure. If now air is released and the point noted where the maximum oscillation of the mercury occurs, one may read approximately the disastolic pressure. The difference between these two readings is, of course, the pulse pressure.

An excellent and reasonable instrument is shown in the illustration (Fig. IV).

Physiologically there are wide fluctuations in the blood pressure. It is least lying down and greatest standing up. Sleep, exercise, food, drink, psychic factors, etc., may alter the blood pressure gradually or suddenly. However, increase of pressure, unless pathological, is not maintained after the particular stimulus has ceased to act.

Undoubtedly the greatest blood pressure values are to be seen in cases of chronic interstitial nephritis where the systolic pressure reaches 270 mm. of mercury or even higher. Not all diseases of the kidney, however, cause increased blood pressure. It is frequently absent in the toxic nephritis cases and in those caused by certain of the infectious diseases. However, in primary acute Bright's disease, which is probably infectious in character, and in the nephritis secondary to scarlet fever, there is practically always an increase in the arterial pressure. This rise may amount to fifty millimeters of mercury within forty-eight hours of the onset of the disease.

Certain forms of arteriosclerosis cause a permanent increase in blood pressure and are accompanied by heart hypertrophy. Such are especially cases of sclerosis of the first part of the aorta and extensive sclerosis of the splanchnic vessels. In uncomplicated arteriosclerosis, only a small proportion of patients show increased blood pressure. When the elasticity of the arteries is diminished, they offer a greater resistance to dilating forces, but once having been dilated, they do

not so easily recover their original size. The rigidity of certain areas may be neutralized by dilatation of other areas. But the splanchnic arteries are of such paramount importance in the regulation of the peripheral resistance that disease in them renders it difficult or impossible for dilatation in other parts of the body to be sufficient to compensate for the splanchnic contraction. (Fig. VI).

Elliott recently in an interesting comparison of the blood pressure in pure arteriosclerosis and in chronic nephritis arrived at the following conclusions: (1) The ordinary clinical type of arteriosclerosis is not necessarily accompanied by high blood pressure; (2) Where high blood pressure is met with in arteriosclerosis, it points to the existence either of associated renal disease, or of sclerosis of the splanchnic vessels and of the aorta above the diaphragm—or both; (3) If we can exclude the renal disease (chronic), splanchnic or aortic sclerosis is to be suspected.

If it were not for the so-called tone of the whole vascular area the heart could not maintain the circulation. This tone is maintained by the contraction of the involuntary muscle

in the vessels. With the exception of the arteries of the brain and of the lungs, there are both vasoconstrictor and vasodilator fibres from the sympathetic nervous system to the smooth muscle fibres of the arteries. The splanchnic area is relatively poorly supplied with dilator fibres. The continuous constrictor impulses sent out from the sympathetic ganglia along the dorsal spine to the arteries keeps the vessels in a state of constriction sufficient to offer enough resistance to the blood flow to facilitate the work of the heart without placing, for any prolonged period, a great strain on it.

It is, therefore, conceivable that an increase in blood pressure may come about in two ways: (1) by stimulation of the constrictor centre (or centres?); (2) by direct action on the muscle cells in the arterial walls. It is believed that the active principle from the medulla of the adrenal gland, adrenalin, is responsible for the maintenance of the arterial tone. It has been found that at various places in the body there are collections of cells known as chromaffin cells, which apparently have an internal secretion analogous

to, if not the same as, the active principle of the adrenal gland. A group of these cells discovered in the heart, has been found to be much hypertrophied in a case of chronic interstitial nephritis accompanied with increased blood pressure and heart hypertrophy.

Clinically, we know that adrenalin causes a rise in the bloodpressure. Experimentally, as we shall discuss later, adrenalin not only is able to cause a rise in blood pressure but also a degeneration of the muscle layer with consequent production of lesions resembling to some extent those of arteriosclerosis in man.

While it must not be forgotten that, given an equal peripheral resistance, a rapid heart will cause the blood pressure to rise, nevertheless this condition usually does not last long. Practically all cases of permanent high tension are due to increase of the peripheral resistance.

CHAPTER III.

PATHOLOGY.

The whole subject of the pathology of arteriosclerosis has been much enriched by the study of the experimental lesions produced by various drugs and micro-organisms upon the aortas of rabbits. Simple atheroma must not be confused with the lesions of arteriosclerosis. The small whitish or yellowish plaques so frequently seen on the aorta and its branches, may occur at any age, and have seemingly no great significance. Such places may grow to the size of a dime or more, and even become eroded. They represent fatty degeneration of the superficial endothelium that at times has no demonstrable cause, at times follows in the course of various diseases, and undoubtedly is due to disturbances of nutrition in the intima. Except for the danger of clot formation on the uneven or eroded spot, these places are of no especial significance, and are not to be confused with the atheroma of nodular sclerosis.

The lesions of true arteriosclerosis are of a different character. It has been customary to differentiate three types, (1) the focal or nodular; (2) the diffuse; (3) the senile. It seems of no great value to make a separate division of the senile form but for convenience of description it will be done. The retrogressive changes of advancing years cannot be rightly termed disease, and the fact even that a man of forty years may have the hard arteries of a man of eighty may mean only that the tissue of the former was poor, the tubing wore out early. Our parents determine the kind of tissue that we shall have as our inheritance. The arteries are elastic tubes capable of much stretching and abuse. In the aorta and large branches there is much elastic tissue and little muscle. When the vessels reach the organs, they are found to be structurally changed in that there is in them a relatively small amount of elastic tissue but a great deal of smooth muscle. This is a provision of nature to increase or decrease the supply at any one point.

The aorta and the large branches are distributing tubes. There is no necessity for

great changes in the capacity of the area. On the contrary, the feed pipes, the actual irrigators, must have some mechanism by which they may flood or curtail the supply of blood to the part. It is after all in the arterioles and smaller arteries, those with considerable muscle fibre that the lesions of arteriosclerosis do the most damage. A point to be emphasized is that the whole arterial system is rarely, if ever, attacked uniformly. That is, there may be a marked degree of sclerosis in the aorta and coronary arteries with very little, if any, change in the radials. On the contrary, a few peripheral arteries only may be the seat of disease. It is not possible to judge the state of the whole arterial system by the stage of the lesion in any one artery, but on the whole an undue thickening of the radial indicates analogous changes in the mesenteric artery and the aorta.

As the body ages, certain changes take place in the arteries leading to thickening and inelasticity of their walls. This is a normal change, and in estimating the palpable thickening of an artery, such as the radial, the age of the individual must always be considered.

Thayer and Fabyan in an examination of the radial artery from birth to old age found that in general the artery strengthens itself as more strain is thrown upon it, by new elastica in the intima, and connective tissue in the media and adventitia. Up to the third decade there is only a strengthening of the media and adventitia. During the third and fourth decades there is also distinct connective tissue thickening in the intima. "In other words, the strain has begun to tell upon the vessel wall, and the yielding tube fortifies itself by the connective tissue thickening of the intima and to a lesser extent of the media." By the fifth decade the connective tissue deposits in the intima are marked, there is an increase of fibrous tissue upon the medial side of the intima and, in lesser degree, throughout the media. The vessel can now be felt as a uniform tube. "Finally, in these sclerotic vessels, degenerative changes set in, which are somewhat different from those seen in the larger arteries, consisting as they do, of local areas of coagulation necrosis with calcification, especially marked in the deep layers of the connective tissue thickenings

of the intima, and in the muscle fibres of the media, particularly opposite these points. These changes may.....go on to actual bone formation." The mesenteric artery differs in some respects from the radial, but in the main the changes brought about by age are the same. Thayer and Fabyan note two striking points of difference: "(1) Calcification is apparently much less frequent than in the radials. (2) In several cases plaques were seen with fatty softening of the deeper layers of the intima and superficial proliferation—a picture which we have never seen in the radial."

In the aorta the elastic muscular intima thickens progressively with age. Scheel has made very careful measurements of the ascending, the thoracic and abdominal aorta, and the pulmonary artery. He found that from birth to 60 years the aorta became progressively wider and lost its elasticity. The pulmonary artery changed little if at all after 30–40 years and where before it was wider than the aorta, it now was found to be smaller. In chronic nephritis both were widened. The continuous increase of width and length of

the aorta stands in reverse relationship to the elasticity of its walls. This is the process that later leads to arteriosclerosis.

So far as the anatomical lesions in the aorta and branches are concerned there is much uniformity even though the etiological factors have been diverse. The only difference is one of extent. To Thoma we owe the first careful work on arteriosclerosis. He regarded the lesion in arteriosclerosis as one primarily situated in the media, a lack of resistance in this coat. A rupture here caused a local widening and consequently the blood could not be distributed evenly to the organ which the diseased artery or arteries supplied. Moreover, there was danger of a rupture at the weak spot unless this spot were strengthened and the lumen again made its former size. Nature's method of repair was a hypertrophy of the subintimal connective tissue and the formation of a nodule at that point. The thickening was compensatory, resulting in the establishment of the normal calibre of the vessel. Thoma showed that by injecting an aorta, the subject of such changes, with paraffin at a pressure of about 160 mm.

Hg., these projections disappeared and the muscle bulged externally. He recognized the fact that the character of the artery changed as the years passed, and to this form he gave the name primary arteriosclerosis. To the group of cases caused by various toxic agents, or following peripheral resistance and consequent high pressure, he gave the name secondary arteriosclerosis. This is a useful division. Even in the diffuse form one will not find lesions of the same grade everywhere. The sclerosis is scattered all over the system; indeed there may be parts that show no lesions whatever. Recently these experiments of Thoma have been repeated and results obtained which are not in accord with his findings. For example, Ophuls finds that stretching of an aorta that has not lost its elasticity will also smooth out the atheromatous plates and nodules, and, moreover, that careful examination of cross sections made through areas beneath the now flattened plates, fails to reveal any evidence of weakness or degeneration of muscle. He attempts to explain the discrepancy between his and Thoma's results by supposing that Thoma examined

late stages of the process when the media was diseased; then it could not be told whether or not the lesions in the media were primary.

In syphilis there are very frequently yellowish-white, irregularly shaped plaques on the intima of the ascending aorta. These are slightly raised above the surface. A tendency to aneurysm formation is present and the aorta is always more or less dilated and has not its customary elasticity. Some have described a special form of mesaortitis due to syphilis, but the majority of pathologists believe that this is not possible. However, this question is not definitely settled, as small gummata have been found beneath the intima and the puckered appearance of the internal coat, that is sometimes seen, is suggestive of a syphilitic scar. Quite recently, *Spirochetae pallidae* have been found in the thickened intima of a patient who died from occlusion of the coronary artery. There was no definite history of syphilis but postmortem a scar was found on the prepuce.

In the nodular form of arteriosclerosis there are places on the aorta and its branches where, as a result of disease and consequent

stretching of the media, there follows a circumscribed dilatation of the vessel. This leads to local compensatory connective tissue growth. The two forms, nodular and diffuse, are more often found together.

The pathological changes vary much in their extent when portions of the same vessel or of different vessels are compared. Frequently, no change is visible from the outside, but again it is readily seen that at one or more spots the artery is widened or shows an irregular contour. On cross section no change in the lumen may be found, or it may show here and there places where it is widened. As a rule the diameter of the lumen becomes greater, and increases as the disease progresses. The stretching of the vessel wall is of a progressive character, and some have thought that the changes in the wall itself are in part due to the destruction of the vasa vasorum.

The changes in the nodular variety affect for the most part the aorta and primarily the ascending portion of the arch. Here there are yellowish or yellowish-white flat projections which are found more frequently

around the orifices of the branches, the orifices of the coronary arteries being especially apt to be the seat of sclerosis.

When these spots represent the beginning of true arteriosclerosis there is already a lesion in the media and compensatory changes are going on, the purpose of which is to strengthen the vessel wall. As the process advances in the arteries these areas undergo molecular changes due probably to destructon of the vasa vasorum. A granular *debris* composed of fatty cells, degenerated cells and cholesterin crystals forms the so-called atheromatous abscess. Should the contents be discharged into the lumen of the vessel the atheromatous ulcer results. Beneath this the ingrowth of connective tissue from the media and adventitia is an attempt to mend the weak spot. Should there be no strengthening at such a point, there is great danger of aneurysmal dilatation.

Sclerosis of the radials of such an extent that these arteries are easily palpable, is a different disease from that of the sclerosis in the aorta. The difference may be due possibly to two factors, (1) the structural dif-

ference in the two vessels, and (2) the difference in the sudden strain put on each at every cardiac systole. In the radial artery it is usually the media that is diseased, the origin of the lesion is in the muscle cells, and the middle coat alone is damaged. The intima and adventitia are not essentially involved, but there is frequently thickening of the intima over the diseased media. In the wall of the vessel the changes that occur are fatty degeneration of the muscle cells and later of the elastic fibres, with subsequent deposits of lime salts in the diseased tissue. There may be calcification of the intima alone, but there is rarely a deposit of lime salts in the media without concomitant deposits in the intima. This gives the beaded character to the vessel. The vessel is perceptibly thinned at the sites of these areas and there result many small pouchings, the intima over these areas being much thickened. These are in reality true minute aneurysms, and when such an artery is held against the light it is seen to be transparent.

On the contrary, the nodular aorta found at autopsy is the result of injuries to the in-

FIG. V.

Fig. V. Cross section of a coronary artery, x50 showing nodular sclerosis. Note the heaping up of cells in the intima, the fracture of the elastica, and the destruction of the media beneath the nodule. The primary lesion apparently was in the media. The thickened intima is the effort on the part of nature to heal the breach. At such places as shown here aneurysms may form.

tima. The intimal thickenings may be entirely proliferative and represent chronic inflammatory production of new tissue. Changes in the media do not necessarily accompany such a lesion. There are some, however, who do not believe that in the aorta the primary lesion is in the intima.

The thickening of the media in the small arteries which is due to the proliferation of connective tissue beneath the endothelium, or to the proliferation of the endothelial cells themselves, may cause such narrowing that thrombosis is favored and a distortion of the vessel is apt to occur. However, the overgrowth alone seldom if ever is sufficient to close the lumen of a vessel. The total occlusion is brought about by a slowing of the blood current, and thrombosis which later becomes organized. Such places may become channeled and allow a certain amount of blood to pass along the thrombosed vessel. These conditions are known, the one as endarteritis deformans, the other as endarteritis obliterans.

Now, the lesions of experimental arteriosclerosis produced by various substances in-

jected into the circulation of animals, have enabled us in a way to judge of the early lesions of arteriosclerosis in the human arteries. Following the injection of small and repeated doses of adrenalin over a certain interval of time, changes occur in the arteries of rabbits that are arteriosclerotic in type, the essential lesion being a degeneration of the muscular and elastic tissue of the media, with the consequent production of aneurysms in the vessel. This is analogous to the arteriosclerosis of the radial artery in man. The degenerations in the arteries after experimental lesions are of the nature of fatty metamorphosis, and later proceed to calcification. Adrenalin, barium chloride, digitalin, physostigmin, nicotine, and other substances have been found to exert a selective toxic action on the muscle cells of the middle coat of the aorta.* The most frequent site of disease in these experimental lesions is the thor-

* The infundibular portion of the pituitary body, the portion which is developed from the infundibulum of the brain, posseses an internal secretion which, injected intravenously, causes a marked rise of blood pressure and slowing of the heart beat. So far as I know this active principle of the gland has not been used to produce experimentally the lesions of arteriosclerosis.

acic aorta, and it is there that the most severe changes are seen. While the toxic action is felt in the vessels all over the body, the lesions are as a rule scattered and small. The thoracic aorta stands the brunt of the high pressure, and this combined with the toxic action of the drug or drugs, results in the formation of a fusiform aneurysmal dilatation which stops at the diaphragmatic opening. The aortic opening in the diaphragm seems to act as a flood gate, allowing only a certain amount of blood to flow through, and thus the abdominal aorta is protected to a great extent from the deleterious effects of increased pressure. Focal degenerative lesions are, however, found in the abdominal aorta.

Changes somewhat analogous to those found in the human aorta as the result of intimal proliferations, are produced in animals by the toxins of the typhoid bacillus and streptococcus. The changes caused by these toxins are proliferations of cells in the intima and subintimal tissues, and a breaking up of the internal elastic lamina into several parallel layers that stretch themselves between the

proliferating cells. The diphtheria toxin on the contrary produces a lesion more like that produced by adrenalin. All pathologists are not agreed as to whether these experimental lesions are truly arteriosclerotic or not; the general trend of opinion seems to be that sufficient work thus far has been done to afford strong support to Thoma's views.

The changes in the intima constitute the effort on the part of nature to repair a defect in the vessel wall which is to compensate for the weakened media and the widened lumen. This applies only to the diffuse type, not to the condition analogous to that produced by the toxin of the typhoid bacillus, for example.

When an artery loses its elasticity and begins to have connective tissue deposited in its walls, the pressure of the blood stretches the vessel now no longer capable of retracting when the pulse wave has passed, and in consequence the artery is actually lengthened. This necessarily causes a tortuosity of the vessel which can be easily seen in the temporals, brachials, radials, and other arteries just beneath the skin.

The exact mechanism of increase in blood pressure is not satisfactorily explained. With the exception of the vessels in the brain and lungs, the smaller arteries are supplied with vasoconstrictor and vasodilator nerve fibres from the sympathetic nervous system. Normally when an organ is actively functioning the vessels are widely dilated and the flow of blood is rapid. Psychic influences of various kinds have a marked effect on vasomotor control of parts of the body.

It is conceivable that in one section of the body the vessels may be markedly contracted; but if there is dilatation in some other part there will be no increased work on the part of the heart, and there need be no rise of blood pressure. The vascular system, while likened to a system of rubber tubes, must be thought of as a very live system, every subsystem having the property of separate control.

For blood tension to be raised all over the body there must be one of two causes ;either the blood must be more viscous, or the conditions over the body must favor the generalized contraction of a large capillary area. The

usual cause of such a condition is the presence in the blood of some poisonous substance.

It must be borne in mind that the great splanchnic area is capable of holding all the blood in the body, and in respect of its liablity to arteriosclerosis, it is second only to the aorta and coronary arteries. The enormous area of the skin vessels could probably contain most of the blood. The fact that the blood is distributed over the body depends on the tone of the vasoconstrictor centre. The fact that the vessels in the splanchnic area are frequently attacked by sclerotic changes means as a rule increase of work for the heart. The resistance offered to the passage of the blood means that for the blood to travel at the same rate that it did before the resistance set in, more power must be expended in its propulsion. In other words, the heart must gradually become accustomed to the changed conditions and as a result of increased work the muscle hypertrophies.

In diffuse arteriosclerosis the heart is always hypertrophied. This is a result, not a cause of the condition. In the pure type, there is hypertrophy only of the left ventricle with-

FIG. VI.

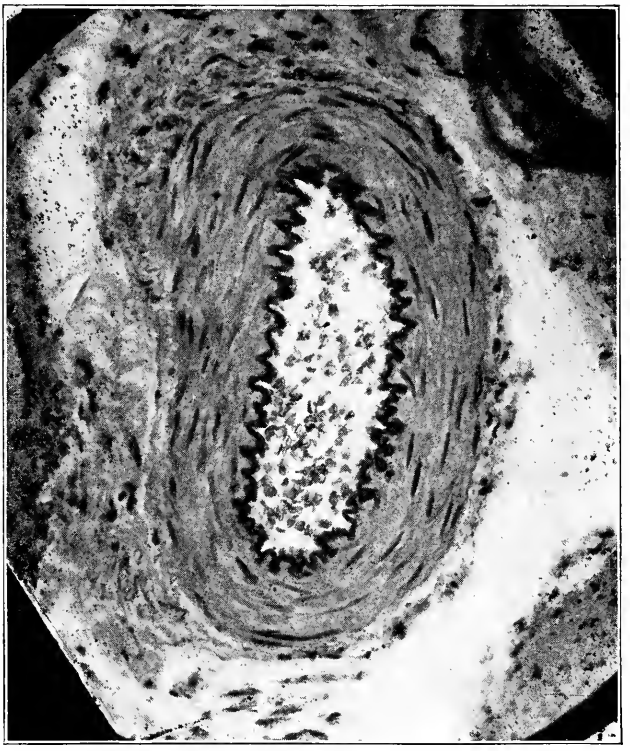

Fig. VI. Cross section of a small artery in the mesentery. Note that vessel appears capable of being much widened. The internal elastic lamina is thrown into folds somewhat resembling the convolutions of the brain. Note also that the middle coat of the artery is composed almost entirely of muscle. The enormous numbers of such vessels in the mesentery and intestines explains the ability of the splanchnic area to accomodate the greater part of the blood in the body. Universal constriction of these vessels would naturally render the intestines anemic. The vasomotor control of these vessels plays an important role in the distribution of the blood. Small arteries in the skin and in other organs, except the brain and lungs, have a similar function. Highly magnified.

out dilatation of the chamber. The muscle fibres are increased in number and size, and there are frequently areas of fibrous myocarditis due to necrosis caused by insufficient nutrition of the muscle. In these cases the coronary arteries share in the generalized arteriosclerotic process. The openings of the arteries behind the semilunar valves may be very small. There is often thickening and puckering of the aortic valves and of the anterior leaflet of the mitral valves leading at times to actual insufficiency of these orifices. Later when the heart begins to weaken there is dilatation of the chambers and loud murmurs result, caused by the inability of the nondistensible valves to close the dilated orifices. Until the compensation is established, it is impossible to say whether or not true insufficiency is present.

In the so-called senile type of arteriosclerosis, a retrogressive change and not what is here called true arteriosclerosis, the arteries may be so lined with deposits of calcareous matter that they appear as pipe stems or they may be tortuous. They feel hard and absolutely nondistensible. At times,

no pulse wave can be felt. When the calcification is not so diffuse, the artery is beaded.

The larger arteries such as the brachials and femorals are most affected. The walls become thinned and show cracks, and areas apparently, but not actually, denuded of intima. White thrombi may even be deposited on these areas and atheromatous ulcers are frequent in such arteries. The danger of an embolus plugging one of the smaller arteries is great, and should the thrombi be on the carotid arteries, hemiplegia may result from cerebral embolism. On microscopical examination of the arteries there is seen extreme degeneration of all the coats, the degeneration of the media leading almost to an obliteration of that coat. On seeing arteries such as these one wonders how the circulation could have been maintained, and the organs nourished. Senile atrophy of the liver and kidneys naturally goes hand in hand with such arterial changes.

There is as a rule no increase in arterial tension; on the contrary the pressure is apt to be low. This is readily understood when the heart is seen. This is small, the

muscle much thinned, flabby and of a brownish tint, the so-called "brown atrophy". Microscopically there is seen to be much fragmentation of the fibres with a marked increase in the brown pigment granules that surround the cell nuclei. Cases are seen, however, in which blood pressure increases as the patient grows older. The hearts in such cases are more or less hypertrophied and show extensive areas of fibroid myocarditis.

There are many cases of arteriosclerosis that lead to definite interference with the closure of the valves of the heart, particularly the mitral and aortic. It has been said above that puckerings of the valves frequently occur. This arteriosclerotic endocarditis at times leads to very definite heart lesions, chiefly mitral insufficiency and aortic insufficiency with murmurs of a stenotic character at the base. There is rarely true aortic stenosis, however. The murmur is caused by the passage of the blood over the roughened valves and into the dilated aorta. Aortic stenosis is one of the rarest of the valvular lesions, and should be diagnosed only when all factors, includng the typical pulse tracings, are taken into consideration.

The kidneys as a rule show extensive sclerosis. A very markedly contracted kidney from which the capsule strips with difficulty may be present, portions of the cortex of the kidney adhering to the capsule. This form is seen for the most part in chronic nephritis, where it may be impossible to say whether the renal or the heart condition was primary. Again, the kidneys may be increased in size, the capsules slightly adherent, the surfaces a little rough. Such organs frequently present atrophic depressed areas, deep red in color. In both types of kidney the consistence is much increased. On microscopical examination, there is widespread deposit of fibrous tissue throughout the organ. Many of the glomeruli are represented only by empty spaces; those which are seen are small and contain much increased fibrous tissue. The new tissue surrounds the tubules; these are compressed and the tubule cells are atrophied, and the arteries show the changes described within.

ARTERIOSCLEROSIS OF THE PULMONARY ARTERIES is an exceedingly rare affection and may occur independently of disease of the

greater circulation. The cases in the literature (four in number) were characterized by wide-spread thickening of the pulmonary arteries with marked hypertrophy of the right ventricle. In two of the cases, no changes in the bronchial arteries or in the pulmonary veins were demonstrable. Three of the cases occurred in persons younger than thirty five years.

Sclerosis of the veins—phlebosclerosis—not infrequently occurs with arteriosclerosis. It is seen in those cases characterized by increased blood pressure. Such increased pressure in the veins is due for example to cirrhosis of the liver which affects the portal circulation, or to mitral stenosis which affects the pulmonary veins. The affected vessels are usually dilated. The intima shows compensatory thickening especially where the media is thinned. Occasionally hyaline degeneration or calcification of the new-formed tissue is seen. "Without existing arteriosclerosis the peripheral veins may be sclerotic, usually in conditions of debility, but not infrequently in young persons" (Osler).

In many cases of arteriosclerosis, the pathological changes are not confined to the arteries but are found in the veins as well as in the capillaries. Such cases could be called angiosclerosis.

CHAPTER IV.

ETIOLOGY.

The causes of arteriosclerosis are many and varied. No two of us have the same resisting power towards poisons that circulate in the blood. Some go through life exposed to all the infectious diseases without ever becoming infected, while others fall easy victims to every disease that comes no matter how careful they may be, and it is quite the same in regard to the resistance of the arterial tissues. If the tubing is of first class quality and the individual does not place too much strain on it, he may live to the Biblical three-score years and ten, and possess arteries that have undergone such slight changes that they are not palpable. Such a person is, however, the exception. On the other hand if the tissue is of poor quality, even the ordinary wear and tear of living causes early changes in the vessels, and a person of forty may have hard arteries.

We have described in the previous chapter the changes which normally occur in the arte-

ries as age advances. An artery that is normal for a man of fifty years would be distinctly abnormal for a boy of fifteen.

Two broad divisions of arteriosclerosis may be made: (1) Congenital, or the result of inherited tendency; (2) Acquired. Of these, the acquired group is by far the larger and more important.

CONGENITAL FORM—When Dr. O. W. Holmes was asked how to live to the age of seventy, he replied that a man should begin to pick his ancestors one hundred years before he was born. Our parents determine the character of the tissues with which we start in life and this determines our general resistance. We might properly speak of congenital arteriosclerosis where the affected individual had poor arterial tissue with which to begin life, for that, in a sense, is a congenital defect, and arterial tissue that is bad is prone to disease.

Arteriosclerosis may occur in infants. Cases have been reported of calcification of the arteries in infants and children. The arteriosclerosis may occur without nephritis or rise of blood pressure. Cerebral hemorrhage in a child of two years has been seen. Heredity

in these cases plays a most important role. In many of the reported cases there was no question of congenital syphilis. Aneurysms, single or multiple, have been found in the arteries of children, and even the pulmonary artery may show sclerotic canges.

Acquired form—All the cases that are usually seen belong in this group. The cases of the previous group are very rare and clinically are not of very great importance.

Hypertension holds first place as a cause of arteriosclerosis. With every systole of the heart, blood is forced out into the arterial system against a certain amount of resistance represented by the tonicity of the capillary area, and the amount of cohesion between the viscous blood and the walls of the arterioles. When a dilatation of the capillaries over any large area takes place, the blood pressure falls, providing there is no compensatory contraction in other areas to make up for the decreased resistance in the dilated vessels. The viscosity of the blood, as such, probably has very little effect on the resistance to the flow. With the systole of the heart there is a sudden dilatation of the

arch of the aorta, and a wave of expansion follows, which is transmitted to the periphery and is lost only in the capillaries.

The blood pressure is constantly changing. Physiologically there are relatively wide variations in the pressure in a perfectly normal individual. There are some persons who have hypotension, a blood pressure much below the normal. Such persons have usually small hearts, small aortas, and they seem to have but little resistance to disease. Many diseases, especially the prolonged fevers, diminish markedly the blood pressure. Whether the hypertension is the cause of the structural changes that are found in the walls of the vessels, or is the result of the diminished area of the arterial tree through which the same amount of blood has to be driven as before the vessel walls became narrowed, is still disputed. As has been stated, experimental evidence would tend to place the initial blame upon the poisons circulating in the blood, which first damage the vessel walls. The subsequent changes then produce thickening and inelasticity. Some think (Allbutt) that the hypertension is primary. There are cases seen clinically

that lend support to this view. Not infrequently individuals in middle life begin to show increase of arterial blood pressure without discoverable cause. It is probable, however, that such cases are those of beginning nephritis where the urine is perfectly normal, as far as chemical examination reveals, but the circulation of some poison in the blood causes a rise of pressure. This is a very interesting group of cases, and more will be said about it later.

No *age* is exempt from the lesions of arteriosclerosis if we consider the two groups. However, the disease is seen for the most part in persons past middle life. The relative frequency with which it is found in the different decades depends on so many factors that it is of no value to tabulate them. As has been stated, arteriosclerosis of all types is an involution process that advances with age. Longevity is a question of the integrity of the arterial tissue, and no one can tell what sort of "vital rubber" (Osler) anyone of us has. However, many with poor tubing may make such use of it that it will outlast good tubing that is badly treated. Unfortunately we

have no way of telling early enough with just what sort of arterial tissue we are starting life.

Sex. There is no doubt that men are far more prone to arterial disease than women are. This is explained by the greater exposure of men to those conditions of life which tend to produce high tension, and so to produce arteriosclerosis, or vice versa. Arteriosclerosis in women is not often seen until after the fiftieth year. Cases of the most extreme grade of pipe stem arteries are, however, seen in old women, and calcified arteries are not hard to find among the inmates of an old woman's home.

Race. The most beautiful examples of arteriosclerosis in this country are seen in the negro. Not only is this disease more frequent in the black race, but the age of onset is much earlier than in the Caucasian. The accidents of arteriosclerosis, viz. aneurysm, cerebral hemorrhage, etc., are more common among the negro males. The etiological factors that are most often found in the history are the prevalence of syphilis and hard physical labor.

Occupation. Certain occupations have a distinct causal relationship to arteriosclerosis; among such are particularly those entailing prolonged muscular exercise, especially if much lifting is necessary. Everyone is familiar with the phenomena accompanying the exertion of lifting. The breath is drawn in, the glottis is closed, and the muscles of the chest wall are held rigidly while the exertion lasts. This causes a great increase in blood pressure, and constant repetition of this will produce permanent high tension. In hospitals, the stevedores as a class have marked arteriosclerosis, and, almost without exception, they are comparatively young men. Occupations that are accompanied with prolonged mental strain, such as now occur to the heads of large manufacturing and financial institutions, also predispose to early arterial changes. Psychic activity, especially when it is accompanied by worry, is a potent factor in the production of the increased blood pressure which is the chief factor in producing arterial disease. There are, however, men who seem not to be harmed by the constant

wear and tear of our modern life. These are the exceptions.

Workers in factories where paint is made and the ingredients hand mixed, are prone to develop arteriosclerosis early in life. It has been found that the laborers most apt to be victims of lead intoxication are those who are careless in their habits of cleanliness, particularly in regard to the fingernails. The continuous absorption of lead into the system, brings about a condition of hypertension that has its inevitable results.

The fact is that any occupation which entails either the absorption of toxic substances, or prolonged muscular labor, will hasten markedly the onset of arterial disease.

Infectious diseases. As more study has been given to the arteries in persons who have died of the acute infectious diseases, more has come to light concerning the effects of the toxins of these diseases on the vessel walls. In the arteries of children who have died of measles, scarlet fever, diphtheria, cerebrospinal meningitis, etc., degenerative changes in the arteries occur, modified only by the length of time that the toxins have acted.

Thayer has shown that the arteries of those who have passed through an attack of moderately severe or severe typhoid fever are as a rule more readily palpable than are the vessels of persons of corresponding years who have never had the disease. Clinically the typhoid toxin appears to cause the early production of arteriosclerosis. The changes in the arteries occur for the most part, and always earlier, in the peripheral arteries, and the media is chiefly affected. Minute yellowish patches are found on the aorta, carotids, and coronaries. In persons who have passed through an attack of one of the fevers, and have later died from some other cause, regenerative changes are sometimes found to have taken place in the arteries, consisting of an ingrowth of elastic fibres from the intact adventitia to the diseased media.

Syphilis. This is one of the most important of the etiological factors in the production of arteriosclerosis. Acute aortitis affecting the ascending and transverse portions of the arch of the aorta is very commonly seen, and the irregular, scattered, slightly raised, yellowish-white patches of sclerosis in

the arch which are found years after the syphilitic lesion, are considered by some to be very characteristic of syphilis. A mesaortitis also occurs that is frequently a *locus minoris resistentiae* where an aneurysm forms. In fact, it is claimed (Osler) that all aneurysms occurring in persons under thirty years of age are due to syphilitic aortitis. In the late stages of syphilis the arterial lesions may be of a diffuse character.

Chronic drug intoxications. Lead, tobacco, and according to some, tea and coffee, are to be classed as causal factors in the production of arteriosclerosis. Certain it is that all these substances have a tendency to raise the arterial pressure, but whether the drug itself causes first a degeneration, and later hypertension results, or vice versa, is not yet positively known. We have just mentioned that lead particularly has a marked effect in producing arterial lesions. Other drugs as adrenalin, barium chloride, physostigmin, etc., while producing experimental arteriosclerosis, hardly could produce the disease in man. Alcohol has been blamed or much, and as an etiological factor in the

production of arteriosclerosis formerly was accorded a first place. More recently much doubt has been thrown on this supposition by the work of Cabot, who showed that the mere drinking of even large quantities of spirits had no effect in producing arterial disease. Nevertheless it is maintained by most clinicians that alcohol is one of the most common causes of arterial disease, and with this opinion we agree. Just what role tobacco plays is difficult to say. My own opinion is, that of itself when used in moderation, it has no ill effects. However as tobacco is a drug that raises markedly the blood pressure, excessive use must be held responsible for the production of arteriosclerosis. It is difficult to separate its effects from those produced by eating and drinking.

Over-eating. There can be no doubt but that the constant overloading of the stomach with rich or difficultly digestible food, is responsible for a large number of cases of arteriosclerosis. Everyone must have noted the increase in force and volume of the heart beat after the ingestion of a large meal. The constant repetition of such processes con-

ceivably can lead to damage to the vessel walls through hypertension. In how far the toxins absorbed from the intestinal tract are responsible for the arterial disease, it is not possible to say. However, they probably do play a certain etiological role, it may be the whole role.

MENTAL STRAIN. Worry kills more people than work. The high pressure under which so many of our people now work is responsible for a not inconsiderable number of cases of arteriosclerosis. The activities of the modern life with its multitude of cares and worries bring many a young head to the grave.

MUSCULAR OVERWORK. This is to be reckoned with as an etiological factor. One sees it especially among the laboring class in both whites and negroes. Possibly other factors as alcohol and coarse heavy food contribute to the early arterial degeneration. Hypertrophy of the heart occurs in athletes, and statistics gathered among the oarsmen especially, show a relatively high mortality at the different decades traceable to the high tension produced while in training. This

question deserves more consideration than has been accorded it.

Renal disease. Chronic disease of the kidneys is one of the surest producers of increased tension; in fact, some see in high tension, even without demonstrable kidney lesions, the earliest sign of a chronic progressive nephritis. There are many, however, who hold that there may be exceedingly high blood pressures without kidney disease. It is possible to divide the cases into two groups that we may call, (1) primary, (2) secondary. By the primary renal disease is meant the group of cases where the kidney disease undoubtedly antedates, by a shorter or longer time, the development of the arteriosclerosis; in other words the arterial disease appears to be caused by the kidney disease.

By the secondary renal disease is meant the group of cases, possibly a small group, where the arterial disease leads to the formation of the kidney lesions. Where the first group occurs for the most part in comparatively young persons, the second group is the result of involuntionary processes due to advanced age.

We have learned that however careful analysis of the urine may be, we can not be sure of the pathological state of the kidney which secretes the urine. Too often so-called normal urine will be secreted by a badly diseased kidney, whereas a urine which contains considerable albumen and many casts may be secreted by a kidney almost perfectly healthy, the lesions being only of a transient and trivial nature.

Too much must not be expected of any one special method of examination; the whole individual must be viewed from every standpoint.

CHAPTER V.

SYMPTOMS AND PHYSICAL SIGNS.

GENERAL—As involution processes are physiological, as has been described (vide infra), arteriosclerosis may assume an advanced grade and run its course devoid of symptoms referable to diseased arteries. It is doubtful if the sclerosis itself could produce symptoms, except in cases later to be described, were it not that the organs supplied by the diseased arteries suffer from an insufficient blood supply and the symptoms then become a part of the symptom complex of any or all the affected organs.

There are cases, however, in comparatively young persons where a combination of certain ill-defined symptoms gives a clue to the underlying pathological processes. These symptoms of early arteriosclerosis are the result of slight and variable disturbances in the circulation of the various organs. Normally there are frequent changes in the blood pressure in the organs but the vasomotor control of normal elastic vessels is so perfect

that no symptoms are noted by the individual. When the arteries are sclerosed, they are less elastic and the blood supply is, therefore, less easily regulated. At times symptoms occur only after effort. The patient may tire more readily than he should for a given amount of mental or bodily exercise; he is weary and depressed, and occasionally there is noted an unusual intolerance of alcohol or tobacco. Vertigo is common especially on rising in the morning or in suddenly changing from a sitting to a standing position. There may be dull headache that the accurate fitting of glasses does not alleviate. Unusual irritability or somnolency with a disinclination to commence a new task may be present. Sometimes the effort of concentrating the attention is sufficient to increase the headache. This has been called "the sign of the painful thought". Numbness and tingling in the hands, feet, arms, or legs are also complained of, and neuralgias, not following the course of the nerves but of the arteries, also occur. It is important to remember that the train of symptoms resembling neurasthenia in a person over forty-five

years old may be incipient arteriosclerosis. This tardy neurasthenia frequently accompanies cancer, tuberculosis, diabetes and incipient general paralysis, as well as incipient arteriosclerosis.

Bleeding from the nose, epistaxis, taking place frequently in a middle-aged person, sometimes is an early symptom. The bleeding may be profuse but is rarely so large as to be positively harmful. In fact, it may do much good in relieving tension. Slight oedema of the ankles and legs is seen. Dyspnoea on slight exertion is not uncommon. Dyspeptic symptoms are not infrequent, pyrosis (heartburn), a feeling of fulness after meals with belching or a feeling of weight in the epigastrium. The dyspeptic symptoms may be so marked that one might almost speak of a variety of arteriosclerosis, the dyspeptic type. For quite a while before any symptoms that would definitely fix the case as one of undoubted arteriosclerosis, the patient complains that foods which previously were digested with no difficulty now give him gastric distress. The examination of the stomach contents of a patient presenting gastric symp-

toms reveals usually a subacidity. The total acidity measured after the Ewald test meal may be only 20 and the free HCl may be absent. Attention has been called to an unnatural pallor of the face in early arteriosclerosis. Progressive emaciation is sometimes seen in cases of arteriosclerosis and may be the only symptom of which the patient complains.

Hypertension—Not all cases of arteriosclerosis are accompanied by increased arterial tension. As has been stated in a previous chapter, the blood pressure in the arterial system depends chiefly on two factors, viz.; the degree of peripheral (capillary) resistance and the force of the ventricular contraction. The highest arterial pressures recorded with the sphygmomanometer occur not in pure arteriosclerosis but in cases where there is concomitant chronic interstitial disease of the kidneys. When this is found there is always arteriosclerosis more or less marked. In cases where the arteries are so sclerosed that they feel like pipe stems there may be an actual decrease in the blood pressure. Hence the clinical measuring of the

FiG. VII.

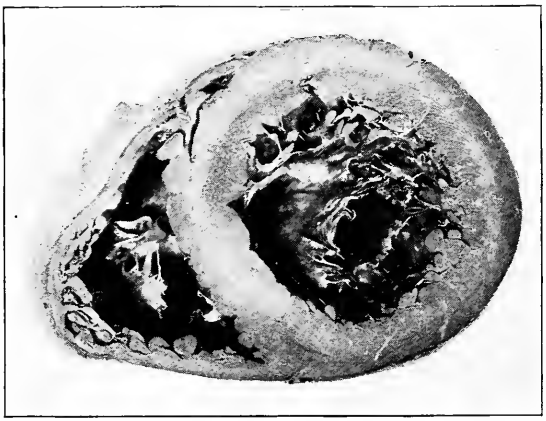

Fig. VII. Enormous hypertrophy of left ventricle due to prolonged increased peripheral resistance. Note that the whole anterior surface of the heart is occupied by the left ventricle. The right ventricle does not appear to be much affected. A case of chronic nephritis. One-half normal size.

pressure in the brachial artery alone is not sufficient for a diagnosis of arteriosclerosis. A persistent high blood pressure even with normal urinary findings is not a sign of arteriosclerosis but of incipient chronic nephritis. The high tension later will lead to the production of sclerosis of the arteries but in these cases the kidney is primarily at fault.

THE HEART—When the arterial tree becomes narrowed and the resistance offered to the flow of blood thereby is increased, more muscular work is required of the left ventricle and according to the general laws which govern muscles the ventricle hypertrophies. There is an actual increase in number of fibres as well as an increase in the size of the individual fibres. Some of the best examples of simple hypertrophy of the left ventricle are found under such circumstances. The chambers as a rule do not dilate until the resistance becomes greater than the contraction can overcome, when symptoms of broken compensation of the heart take place. The hypertrophy of the left ventricle brings more of this portion of the heart towards the anterior chest wall. The enlargement

is towards the left also, consequently the apex beat is found below and to the left of its usual site, even an inch or more beyond the nipple line. The impulse is heaving, pushing the palpating hand forcibly up from the chest wall. The visible area of pulsation may occupy three interspaces and the precordium is seen to heave with every systole. On auscultation the second sound at the aortic cartilage is ringing, clear, and accentuated. Not infrequently, too, the first sound is loud and booming but has a curious muffled sound that may even be of a murmurish quality. The leaflets of the mitral valve may be the seat of sclerosis, the edges slightly thickened and not quite approximating causing a definite murmur with every systole. This murmur may be transmitted out into the axilla and heard at the inferior angle of the left scapula.

Palpable Arteries—Not every artery that can be felt is the subject of arteriosclerosis, and, as has been stated, palpable arteries being more or less a condition of advancing years, judgment as to whether the artery is pathologically or physiologically thickened

may be a matter of individual opinion. A radial artery that lies close to the lower end of the radius and can actually be seen to pulsate when the hand is held slightly extended on the back of the wrist, is easily felt but must not, therefore, be considered a sclerosed artery. The radial may be so deeply situated in the wrist of a fat subject that it is difficultly palpable. Yet the two cases just described may have arteries of identical structure, there being no more retrogressive changes in the one than in the other. "Experience is fallacious and judgment difficult".

There is also a right way and a wrong way to palpate the artery, the radial for example. No accurate data can ever be obtained by feeling the artery with one hand. The small, contracted, wiry artery of a chronic nephritic may feel like a pipe stem, but if properly felt the mistake will not be made of considering such an artery an unusually sclerosed one.

To palpate the radial artery, both hands are used. With the middle finger of the right (left) hand the artery is compressed peripherally, that is, nearest the wrist. The blood is then pressed out of the artery with

the middle finger of the left (right) hand, so as to obliterate completely the pulse wave and the two or three inches between the middle fingers are felt with the index fingers. By holding the finger firmly on the artery near the wrist so as to block any wave that may come through the palmar arch by anastomosis with the ulnar artery and by releasing pressure on the proximal middle finger, some idea may be had of the degree of pulse tension. However, no amount of practice can more than approximate the tension and when one is surest that he can tell how many millimeters of pressure there is, he is apt to be farthest wrong when he checks his guess with the sphygmomanometer.

Much may be learned from carefully palpating the peripheral arteries, and, as a rule, the sclerosis of these arteries means general arteriosclerosis although there are many exceptions to this.

Ocular symptoms—These are important but are as a rule not found because not looked for. Not every practitioner is skilled in the use of the ophthalmoscope. Frequently, the disease is recognized first by the ophthal-

mologist. In general, the symptoms are gradual loss of acute vision, and attacks of transient loss of vision. The explanation which has been offered for these phenomena is the contraction in a diseased central artery. In the fundus are seen increased tortuosity of the retinal vessels and their terminal twigs with more or less bending of the vessels at their crossings. The arteries are terminal ones and small patches of retinitis are therefore found. The changes have been divided into (1) Suggestive, (2) Pathognomonic.

Under (1) are (a) Uneven calibre of the vessels,
(b) Undue tortuosity,
(c) Increased distinctness of the central light streak,
(d) An unusually light color of the breadth of the artery.

Under (2) are (a) Changes in size and breadth of the retinal arteries so that they look beaded,
(b) Distinct loss of translucency,

(c) Alternate contractions and dilatations in the veins,

(d) Most important of all, the indentation of the veins by the stiffened arteries.

Moreover, there is the arcus senilis, the fine translucent to opaque circle surrounding the outer portion of the iris. Practically every one with a well marked arcus senilis has arteriosclerosis, but vice versa not every one with even marked arteriosclerosis has an arcus senilis.

NERVOUS SYMPTOMS—The onset of arteriosclerosis is, in the majority of cases, so insidious that certain nervous manifestations due in all probability to disturbances in blood pressure, are present long before the actual sclerosis of the arteries can be felt. These nervous symptoms are at times the sign posts to show us the way to the accurate diagnosis. There may be gradual increase in irritability of temper, inability to sleep, vertigo even extending to transient attacks of unconsciousness. Loss of memory for details and

nervous indigestion may be present. Various paresthesias as numbness, tingling, a sense of coldness or of heat or burning, a sense of stiffness or even actual stiffness or weakness may occur in the arms and legs, more frequently in the legs. The pain complained of may be due to occlusion of an artery, although evidence for this is lacking. It has been thought by some that the pain in angina pectoris might be due to this cause.

Several curious and interesting diseases which have been thought by some to have arteriosclerosis as a basis are accompanied by pain. Such are erythromelalgia, Raynaud's disease, "dead fingers", and intermittent claudication.

There is a group of cases that is characterized by a period of prolonged low fever, the fever never rising as a rule above 100.5 degrees F. to 101 degrees F., but lasting for weeks and even months. Except for a feeling of malaise, slight headache, and possibly slight dyspnoea on exertion, the patient seems to be fairly well. When after careful observation and elimination of all the common causes of prolonged fever, including syphilis

and tuberculosis, one is at a loss to know what the disease is, there may be a possibility that subacute arteriosclerosis may be the cause of the symptoms. There are times when the disease may progress very rapidly. Cases are seen in which the peripheral arteries become hard and stiff a short two years from the onset.

Well developed arteriosclerosis shows four pathognomonic signs: (1) Hypertrophy of the heart, (2) Accentuation of the aortic second sound, (3) Palpable thickening of the arteries, and (4) Heightened blood pressure. However, it must not be inferred that these signs must be present in order to diagnose arteriosclerosis. It has already been said that a very marked degree of thickening with even calcification of the palpable arteries may occur with absolutely no increase of blood pressure, and at autopsy a small flabby heart may be found.

In this connection, the classification of Prof. T. Clifford Allbutt is worthy of extended notice. He divides the causes of arteriosclerosis clinically into three classes: 1. The toxic class—the results of poisons of the

most part of extrinsic origin, chiefly those of certain infections. In some of these diseases, the blood pressures, as for example, in syphilis, are ordinarily unaffected; in others, as in lead poisoning, they are raised. 2. The class he calls hyperpietic,* in which an arteriosclerosis is the consequence of tensile strength, of excessive arterial blood pressure persisting for some years. A considerable example of this class is the arteriosclerosis of granular kidney, but in many cases kidney disease is, clinically speaking, absent. 3. The involutionary class, in which the change depends upon a senile, or quasi-senile degradation. This may be no more than wear and tear, a disposition of all or of certain tissues to premature failure—partly atrophic, partly mechanical—under ordinary stresses; or it also may be toxic, a slow poisoning by the "faltering rheums of age". In ordinary cases of this class the blood pressures for the age of the patient, are not excessive. Although the toxins of the specific fevers, notably typhoid, as stated above, and

* From πιεζω to squeeze, straiten, oppress or distress. Hyperpiesis, therefore, signifies excessive pressure.

influenza, have been shown to produce arteriosclerosis, this, under, favorable circumstances he believes tends to disappear. This has been shown by Wiesel (v. infra).

As the blood pressure is dependent on the resistance offered by the capillaries and arterioles, there are only two ways in which increased pressure can be brought about; either by rendering the blood more viscous, or by the generation of some poison from the food taken into the body which, acting on the vasomotor centre, or directly on the finer vessels, arteriolar or capillary, sets up a constriction over any large area, and mainly in the splanchnic area. In regard to the liability to arteriosclerosis, this area stands second only to the aortic and coronary areas. He believes that arteriosclerosis itself has little effect in raising arterial pressure. Many cases are seen in which with extreme arteriosclerosis there was no rise in blood pressure, and some in which pressures have been rising even long before the appearance of arterial disease. Prof. Allbutt also believes that in the hyperpietic cases the arteries undergo a transient

thickening, which can be removed if the causes can be reached and overcome.

Clinically speaking, then, hyperpietic arteriosclerosis is not a disease but a mechanical result of disease. If the narrowing of the arterioles is brought about by thickening due to arteriosclerosis, then it would seem *a priori* that such obliteration should cause a rise in pressure. Were the vascular system a mere mechanical set of tubes and a pump, this would happen, but other factors of great importance must be taken into consideration besides, the mechanical factors, viz:; chemical and biological factors. Thus, whole parts may be closed and with compensatory dilatation in other parts there would be little or no change in pressure, unless there were hyperpiesis. In established hyperpiesis, we note two conditions in the radial artery, first a comparatively straight vessel with a small diameter; secondly, a larger, more tortuous vessel, "the large leathery artery." In the cases of the first group, hyperpiesis is often more marked, although not appearing so to the examining finger, than in the second class. In view of the difficulty of estimating by

touch alone the amount of hyperpiesis in a contracted hard artery, it is often overlooked until a ruptured vessel in the brain startles us to a realization of our mistake. The "narrow" artery is more dangerous than the tortuous one, for with every change in pressure the passive vessels of the brain must receive blood that under normal conditions would go to other parts of the circulation.

In involutionary sclerosis there is a gradual thickening and tortuosity of the vessel, which, although it may be greater than in the hyperpietic cases, yet is never so dangerous to life. The heart in hyperpiesis hypertrophies and dilates, but such a heart is the result, not an integral part, of the arterial disease.

CHAPTER VI.

SYMPTOMS AND PHYSICAL SIGNS.

(Continued.)

Although arteriosclerosis is a disease which affects the whole arterial system, it nevertheless never reaches the same grade all over the body. The difference in the structure and functions of the various organs determines to great extent the eventual symptomatology. Endarteritis obliterans of a small sized artery in the liver or leg would lead to no marked symptoms as the circulation is so rich that the anastomoses of the blood vessels would soon establish a collateral circulation that would be perfectly competent to sustain the function of the part. Quite different would it be should one of the small arteries of the brain, the lenticulo-striate, for example, which supplies the corpus striatum, become the seat of a thrombosis or embolism caused by arteriosclerosis. The arteries of the brain are terminal arteries and the blood supply would be cut off entirely with a resulting anemic necrosis of the part supplied

by the artery and a loss of function of the part. What would be of no moment in the leg or arm, might prove even fatal in the brain.

The further symptomatology, therefore, of arteriosclerosis depends entirely on the organ or organs most affected by the interference with the blood supply. The following groups may be recognized:

1. Cardiac
2. Renal
3. Abdominal
4. Cerebral
5. Spinal
6. Local vaso-motor effects.

(1) Cardiac—Most cases of arteriosclerosis sooner or later present symptoms referable to the heart. When the organ is hypertrophied and is already working against an enormous peripheral resistance a slight excess of work put upon it may cause a dilatation of the chambers with the resulting broken compensation. There is dyspnoea on slight exertion, possibly some precordial distress, slight oedema of the ankles and lower legs and possibly scanty urine. With proper care,

such a patient may recover but the danger of another break in compensation is enhanced. The next attack is more severe. The oedema is greater, there may be signs of oedema of the lungs, effusions into the serous cavities may occur. The heart shows marked dilatation. There is gallop or canter rhythm and there are loud murmurs at the apex. When a patient is first seen in this stage it may be quite impossible to state whether or not there is true valvular disease of the heart. The muscle is usually diseased in that there is fibroid degeneration of more or less extensive character. This factor causes the heart to lose much of its elasticity and increases the tendency to permanent dilatation. Such cases must be watched before one can say that true valvular insufficiency is not present. The fatal termination of such a case is quite like that of true valvular disease. There is increasing dyspnoea, increasing anasarca, and the patient usually succumbs to oedema of the lungs, drowned in his own secretions.

A very rare complication of the fibroid degeneration of the heart muscle is aneurysm of the heart wall. The apex of the left ven-

tricle is most commonly the site of the aneurysm and rupture occasionally occurs. Such an accident is rapidly fatal. In the arteriosclerotic process which occurs at the root of the aorta, the coronary arteries become involved both at the openings and along the courses of the vessels. A branch or branches or even one artery may become blocked as a result of obliterating endarteritis. The arteries of the heart are terminal vessels and as a rule blocking of them leads to anemic infarcts. These areas become replaced by fibrous tissue which in the gross specimen appears as streaks of whitish or yellowish color in the musculature. Anemic infarcts may not occur. If such is the case, there must be either abnormal anastomotic communications between the otherwise terminal vessels, or the circulation is maintained by means of the vessels of Thebesius. Through arteriosclerosis of the coronary vessels extensive fibrous changes may occur that lead to a myocarditis with its attending symptoms—dyspnoea, irregular and intermittent heart, gallop rhythm, oedema, etc. One of the most distressing and dangerous results

FIG. VIII.

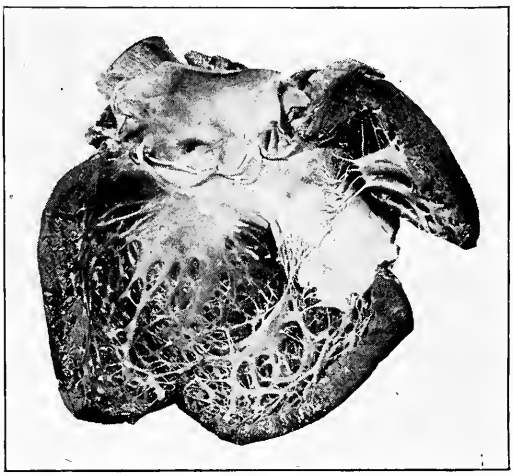

Fig. VIII. Aortic incompetence with hypertrophy and dilatation of left ventricle the result of arteriosclerosis affecting the aortic valves. Note how the valves have been curled, thickened, and shortened, the edges of the valves being a half an inch below the upper points of attachment. The anterior coronary artery is shown, the lumen somewhat narrowed. One-quarter size.

of sclerosis of the coronary arteries and of the root of the aorta is angina pectoris. While in almost every case of angina pectoris there is disease of the coronary arteries, the contrary does not hold true, for most extensive disease, even embolism, of the arteries is frequently found in persons who never suffered any attacks of pain. This symptom group is more common in males than in females and as a rule occurs only in adult life. "In men under thirty-five syphilitic aortitis is an important factor." (Osler).

Since the valuable experiments of Erlanger on Heart Block, considerable attention has been paid to lesions of the Y-shaped bundle of fibres, a bundle arising at the venous orifices in the right auricle and extending to the two ventricles, known also as the auriculo-ventricular bundle of His. Interference with the transmission of impulses through this bundle gives rise to the symptom group known as the Stokes-Adams syndrome,* which

* The bundle of His has been found to be the pathway through which the impulses, that stimulate the heart muscle to contract, reach the ventricles. The mechanism briefly is this. Impulses apparently have their origin at the openings of the inferior and superior cava in the right

is characterized by (a) slow pulse, (b) cerebral attacks,—vertigo, syncope, transient apoplectiform and epileptiform seizures, (c) visible auricular impulses in the veins of the neck. Many of the cases which occur are in elderly people the subjects of arteriosclerosis.

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auricle. Contraction of the heart,as shown by cardiograms and cardioplethysmographs, moves in a wave towards the ventricles. Normally there seem to be several stimuli originating at the venous orifices, all of which,however, do not reach the ventricles. There is, moreover, a definite ratio between the number of stimuli and the actual number of ventricular contractions. These may be 4:1, 3:1, 2:1, 1:1. Thus it is clear that there may be 4, 3, 2, 1 auricular contractions to one ventricular contraction. Now by experimentally compressing the heart of the dog at the auriculo-ventricular ring by means of a special clamp there is marked disturbance in the relation of the auricular and ventricular contractions. This blocking of impulses may be carried on to the extent that the auricles and ventricles beat absolutely independently of one another. Slight blocking may give extra systolic beats heard at the heart but not felt in the pulse.

In Stokes-Adams syndrome we find that lesions of the bundle of His give us much the same phenomena as we can experimentally produce on animals by actual compression of the bundle.

Those interested in further information may find it by consulting articles by Erlanger, Dawson, Hirschfelder, Hewlett, etc., in Jour. Exper. Med., Brit. Med. Jour., Am. Jour. Med. Sc., Jour. Amer. Med. Assoc., Johns Hopkins Hospital Bulletin, etc. All the literature is contained in articles written during the past four years.

There is disease of the bunde of His (auriculo-ventricular bundle) in cases recently carefully examined. G. C. Robinson in a collection of 16 cases found most of them due to gummatous lesions of the bundle, some however, were arteriosclerotic in type. The condition is most interesting and careful autopsies should be made on all those dying with symptoms of Stokes-Adams disease.

Renal—There are those who see in increased arterial tension renal disease always manifest or latent. While it is a valuable observation that persistent high tension is very frequently the warning sign of a beginning chronic nephritis, one cannot say that such is always the case. We frequently see elderly patients in whom there is high tension and some arteriosclerosis of the palpable arteries, but in whose urines repeated examinations fail to reveal the slightest evidence of renal disease. Such kidneys are small and granular, to be sure; they are known as the senile kidney. The connective tissue is increased and there is also throughout the body, evidence of senile atrophy. Such kidneys connot strictly be said to be diseased even

though they may appear much like the kidneys of much younger persons who have died from definite chronic nephritis. However, in a large number of cases of arteriosclerosis renal symptoms appear. There is oedema of the ankles and legs, puffiness of the eyelids, anemia, etc. There may be uremic amauroris, uremic convulsions, or symptoms of mild uremia, such as headache, transient attacks of blindness, dizziness, vertigo, etc. It is not possible always in a given case to decide whether the arterial or the renal disease was the primary one. Clinically the question is not of such great importance, as the end results are practically the same whichever system was first involved, where symptoms of both put in their appearance.

Abdominal or Visceral.—There is an important group of cases to which but little attention has been paid until quite recently. This is the abdominal or visceral type of arteriosclerosis. It has been stated that arteriosclerosis of the splanchnic vessels almost invariably causes high tension. Among others, Janeway has shown that general arteriosclerosis without marked disease of

the splanchnic vessels does not cause as a rule increase of blood pressure.

There are cases in which the brunt of the lesion falls upon the abdominal vessels. Such cases have been called "Angina abdominalis". It has been suggested (Harlow Brooks) that this type of arteriosclerosis may be determined by constant overloading of the stomach with food, especially rich and spiced food. This causes overwork of the special arteries connected with digestion and so leads to sclerosis of the vessels of the stomach, pancreas, and intestines. Personal habits probably influence to great extent the production of this more or less *localized* condition.

The organs supplied by the diseased arteries suffer from changes analogous to those occuring in general or local malnutrition, such as starvation, old age, or local anemias. These changes are atrophy with hemachromatosis (brown atrophy) or fatty infiltration and degeneration. Following the degenerative changes there result connective tissue growth and further limitation of the functioning power of the affected organs.

Pain is a more or less constant symptom of visceral sclerosis. In the early stages there may be only a sense of oppression, of weight, or of actual pressure in the abdomen or pit of the stomach. There may be only recurring attacks of violent abdominal pain accompanied by vomiting. In some cases symptoms of tenderness in the epigastrium, pains in the stomach after eating, vomiting and backache may suggest gastric ulcer. There may be dyspnoea and a sense of anguish accompanied with a rapid and feeble pulse. Hematemesis may make the symptom group even more like ulcer of the stomach, and only the course of the disease with the failure of rigid ulcer treatment and the substitution of treatment directed towards relief of the arterial spasm with resulting betterment, enables one to make a diagnosis. The condition may be present for years and the symptoms only epigastric tenderness with dizziness and sweating on lying down after dinner, as in one of Perutz's patients. The attacks are probably due to spasmodic contraction of the sclerosed intestinal vessels with a resulting local rise in blood pressure. The pains

are probably located in the sympathetic and mesenteric plexuses.

This result of arteriosclerosis is not so uncommon, and by keeping this cause of obscure abdominal pain in mind we are now and then enabled to save a patient from operation.

The following case reported by Neusser will serve as a good example of this so-called angina pectoris gastralgica.

L. G. 47 years old, married, letter carrier, adm. June 12, 1902. The patient had had malaria. At 30 and 40 yrs. he had ischias; suffered much from eructations and when 42 had catarrh of the stomach, which was the beginning of his present illness, and for which he underwent a rigid diet and Carlsbad water cure.

On admission, he complained that when he returned home after work, he suddenly experienced a burning and a feeling of oppression in the stomach, he could not breathe freely, but during the pain had to remain standing and take short, quick breaths. Such an attack lasted about a minute, then he could go on. This sort of

attack was repeated every 8 to 14 days. In summer, he had fewer attacks, in fall they became more frequent and especially frequent and severe in winter. Between attacks he felt quite well, could eat anything, had no eructations, no vomiting. Recently the attacks had become more frequent and more severe. Milder attacks began with a drawing pain in the pit of the stomach and a feeling of fulness in the stomach. As soon as the attack ceased there was some belching and then relief was felt. When the attack came on shortly after eating, there followed troublesome vomiting and then relief. Severe attacks began with burning pain in the stomach located deeply, which radiated outwards over the chest to the manubrium sterni, with a feeling in the larynx as if the parts were being screwed together, violent pain, that extended fromthe neck over both under jaws to the temples, at the same time there followed sweating and salivation now and again and also radiation of the pain to the teeth.

In especially severe attacks the pain was felt between the shoulders as a band around the chest; deep breathing was impossible.

Following the attacks there was great weakness. Actual feelings of impending dissolution the patient never had, likewise no streaming pains in the left or right shoulders, in the arm or below; no dizziness, no palpitation. The pulse showed no abnormality during the four to fifteen minute attacks.

The exciting causes of the attacks recently were many. Not only walking but also lifting, psychic exertions especially at his work, changes of temperature, e. g., leaving a hot room in winter for the outside cold, would bring on attacks. Especially easily were attacks brought on by taking spicy foods, cheese, wine, whiskey. In the intervals he felt well, had a good appetite, the bowels were regular. He was a moderate drinker and smoker. He had had gonorrhoea at 22 years; a soft chancre at 23, no skin eruptions.

P. E.—He is a well built man, the pupillary reaction is prompt, the patellar reflexes are normal. The mucous membranes are somewhat livid, the skin is slightly icteric. There is visible pulsation at the jugular notch; the arteries are not hard. The sphygmomanometer (Basch) reading is 115;

pulse 72; the lungs are normal. The point of maximum impulse of the heart is palpable in 5th interspace, somewhat displaced outwards; the dulness reaches on right to sternal border. The second aortic is ringing; after the sound there is a diastolic murmur which is transmitted downwards as far as the xiphoid. Posteriorly to the left from the spine the diastolic murmur and the second ringing tone are plainly heard.

Diagnosis: Sclerosis of the thoracic aorta and insufficiency of the aortic valve. Angina pectoris coronaria.

On July 26th the patient left the hospital much improved.

CEREBRAL — It has been stated that arteriosclerosis is a general disease yet certain systems of vessels may be affected far more than others and indeed there may be marked sclerosis at one part of the body and none demonstrable at another part.

In advanced sclerosis there may be one or more of a series of accidents due to embolism, thrombosis, or rupture of the vessels. Such conditions as transient hemiplegia, monoplegia or aphasia may occur. The attacks

may come on suddenly and be over in a few minutes; what Allbutt calls "Larval apoplexies". They may last from a few hours up to a day; they are very characteristic. A patient aged 64 years with pipestem radials and tortuous hard temporals would be lying quietly in bed when suddenly he would stiffen, the eyes would become fixed and the breathing cease. In a few seconds consciousness returned, the patient would shake himself, pass his hand over his brow and ask "Where am I? Oh, yes, that's all right". He had as many as thirty of these attacks in twenty-four hours, none of them lasting over one minute. Just what such attacks are due to it is hard to say. Some have attributed them to spasm of the smaller blood vessels of the brain, but there have never been demonstrated in the vessels any constrictor fibres.

There is a well recognized form of dementia caused by arteriosclerosis. In general paralysis of the insane and in senile dementia the blood vessels are always diseased. Milder grades of psychic disturbances are accompanied by such symptoms as mental fatigue, persistent headaches, vertigo, memory

weakness and fainting. Aphasia, periods of excitement and mental confusion occur in some. Later stages are at times accompanied by inclination to fabulate, loss of judgement, disorientation, narrowing of the external interests, episodes of confusion and hallucinatory delirium.

The hemiplegias, monoplegias and paraplegias may occur again and again and last for one or two days. Unless there has been rupture of the vessels there is complete recovery as a rule.

In persons who have arteriosclerosis with high tension attacks of melancholia are seen. There are at the same time fits of depression, insomnia, irritability, fretfulness and a generally marked change in disposition. When the tension is reduced by appropriate treatment these symptoms disappear, to recur when the tension again becomes high. On the contrary attacks of mania are accompanied by low blood preasure. The dizziness and vertigo in cerebral arteriosclerosis are probably due to the stiffness of the vessels which prevents them from following closely the variations of pressure produced by

position, and thus, at times, the brain is deprived of blood and a transient anemia occurs.

In Stokes–Adams syndrome the slow pulse is due as has been stated to some lesion of the auriculo–ventricular muscle bundle in the heart. The epileptiform and apoplectiform attacks which occur are due to the sclerosis of the cerebral vessels. In a case, seen by the writer, of a colored man thirty–two years of age who gave a history of syphilis, the cerebral sclerosis as well as the lesion in the heart was in all probability due to the syphilitic toxin.

Arteriosclerosis of the cerebral vessels is always a serious condition. The greatest danger is from rupture of a blood vessel. Another of the dangers is gradual occlusion of the arteries bringing about necrosis with softening of the brain substance. The latter is more apt to be associated with psychic changes, dementia, etc.; the former, with hemiplegia. It is curious that a small branch of the Sylvian artery, the lenticulostriate, which supplies the corpus striatum, should be the one which most frequently ruptures. Where the motor fibres from the whole

cortex are gathered together in one compact bundle a very small hemorrhage may and does cause very serious effects. A comparatively large hemorrhage in the silent area of the brain may cause few or no symptoms.

Spinal.—It is conceivable that arteriosclerosis of the vessels of the spinal cord might cause symptoms which would be referred to the areas of the cord where the process was most advanced. The lesions would be scattered and consequently the symptoms might be protean in character.

True epileptic convulsions dependent on arteriosclerotic changes are also seen and are not so uncommon.

This is on the whole a rare condition, much less common than arteriosclerosis of the cerebral vessels. Collins and Zabriskie report the following typical case:

"H., a fireman ,51 yrs. old, was in ordinary good health until toward the end of 1902. At that time he noticed that his legs were growing weak and that they tired easily. Later he complained of a jerking sensation in different parts of the lower extremities and at times of sharp pain, which might last from

several minutes to two or three hours. The legs were the seat of a heavy, unwieldly sensation, but there was no numbness or other paresthesia. About the same time he began to have difficulty in holding the urine, a symptom which steadily increased in severity. These symptoms continued until March 1903, i. e., for three months, then he awakened one morning to find that he was unable to stand or walk, and the sphincters of the bowels and bladder relaxed. There was no complaint of pain in the back or legs, no difficulty in moving the arms, in swallowing or in speaking. He says he was able to tell when his lower extremities were touched and he could feel the bed and clothes. He was admitted to the City Hospital three weeks later and the following record was made on April 21, 1903.

The patient was a frail, emaciated man of medium height, who had the appearance of being 55–60 yrs. of age.. He was unable to stand or walk. When he was lying he could flex the thigh and the legs slowly and feebly. There was slight atrophy of the anterior and inner muscles, more of the left than

of the right side. The knee jerks and ankle jerks were absent. Irritation of the soles caused quite a typical Babinski phenomenon. The patient had fair strength in the upper extremities, but the arms tired very soon, he said. The grip was moderate and alike in each hand. The motility of the face, head, and neck was not noticeably impaired. There was no difficulty in swallowing, and articulation was not defective. Tactile sensibility was slightly disordered in the lower extremities, although he could feel contact of the finger, the point of a pin, and the like. Sensibility was not so acute as normal; there was a quantitative dimunition. Sensory perception was not delayed. There was a distinct zone of slight hyperesthesia about as wide as the hand above the femoral trochanters. Above that, sensibility was normal. There was no discernible impairment of thermal sensibility. No part of the body was particularly tender on pressure. A bedsore existed over the sacrum, and there was excoriation of the genitals from constant dribbling of urine.

Examination of the chest showed shallow respiratory movements. The heart was regular, weak, there were no murmurs, the second sound was accentuated. Examination of the abdomen showed that the liver and spleen were palpable, but were not enlarged. The abdominal reflexes, both upper and lower, were sluggish. The patient was slow of speech, likewise apparently of thought. He did not seem to show an adequate interest in his condition, still he was fully oriented and seemed to have a fair memory. His mental reflex was slow. There were indications in the peripheral blood vessels and heart of a moderate degree of general arteriosclerosis. The peripheral vessels, such as the radial, were palpable, the walls thickened, the blood pressure increased.

The patient did not complain of pain while he was in the hospital, a period of four weeks, nor was there any particular change in the patient's symptoms, subjective and objective, during this time. His mental state remained clear until forty-eight hours before death, when he became sleepy, stuporous, and comatose, dying apparently of cardiac weak-

ness, which had set in simultaneously with the clouding of consciousness.''

At autopsy, except for a few small hemorrhages in the posterior horns of the lower dorsal segments on the right side and a similar condition of the left anterior horn, there was nothing noticed. On microscopical examination, there was widespread sclerosis of the vessels of the cord of a marked degree with only slight thickening of the vessels of the brain. There were secondary degenerations of ascending and descending type particularly marked at the ninth dorsal segment. They included portions of all the tracts, the pyramidal tract as well. The symptoms in brief were: (1) Weakness and easily induced fatigue of the legs; (2) peculiar sensations in the lower extermities, described as jerky, numbness, heaviness, and occasionally sharp pain; (3) progressive incontinence of urine; (4) progressive paraplegia.

Local or peripheral—When the arteriosclerosis in the peripheral arteries reaches a stage where endarteritis obliterans supervenes, there is usually no chance for a compensatory or collateral circulation to be estab-

lished. The area supplied by the vessel undergoes dry gangrene. A portion of a toe or finger or a whole foot or hand may shrivel up. It is more common to see the spontaneous amputation take place in the lower extremities. The same effect may be produced by the plugging of a vessel with a thrombus. There may be much pain connected with the sudden blocking, whereas the gradual obliteration of the blood supply of a toe or foot is not as a rule at all painful. The condition is at times revealed more or less accidentally when a patient injures his toe or foot and discovers that there is no sensation to the part and that the wound instead of healing is inclined to grow larger.

Other interesting vasomotor phenomena are frequently connected with arteriosclerosis. Such a one is the curious condition known as Raynaud's disease, a vascular disorder which is divided into three grades of intensity: (1) local syncope, (2) local asphyxia, (3) local or symmetrical gangrene. This is not the place to describe this condition except to say that the condition called "dead fingers" is the most characteristic feature of

the first stage. Chilblains represent the mildest grade of the second stage. The parts are intensely congested and there may be excruciating pain. Anyone who has ever had chilblains knows how painful they can be. The general health is not impaired as a rule, although the attacks are apt to come on when the person is run down. The third stage may vary from a very mild grade, with only small necrotic areas at the tips of the fingers, to extensive multiple gangrene.

Another and very rare condition in which chronic endarteritis was the only constant finding is the disease discribed by S. Weir Mitchell and called by him erythromelalgia (red neuralgia.) This is "A chronic disease in which a part or parts—usually one or more extremities—suffer with pain, flushing, and local fever, made far worse if the parts hang down." (Weir Mitchell).

Probably the most frequently seen result of arteriosclerosis in the leg arteries is the remarkable condition, first described by Charcot, known as intermittent claudication. Persons the subject of this disease are able to walk if they go slowly. If, how-

ever, any attempt be made to hurry the step, there results total disability accompanied at times by considerable cramp-like pain. The condition is much more prone to occur in men than in women, and Hebrews seem more frequently affected. The cause is most probably to be sought in the anemia which results from the narrowing of the channels through which the blood reaches the part. The stiff, much narrowed arteries allow sufficient blood to pass along for the nutrition of the part at rest or in quiet motion. Just as soon as more violent exercise is taken, calling for more blood, an ischemia of the part supervenes, for the stiff vessels cannot accomodate themselves to changes in the necessary vascularity of the part. A rest brings about a gradual return of blood and the function of the part is restored. Pulsation may be totally absent in the dorsal arteries of the feet and when the legs are allowed to hang down there is apt to be deep congestion.

In this connection a curious case reported by Parkes Weber will not be out of place. The patient, a male, aged 42 years complained of cramp-like pains in the sole of the left foot

and calf of the leg occurring after walking for a few minutes and obliging him to rest frequently. When the legs were allowed to hang over the side of the bed, the distal portion of the left foot became red and congested looking. No pulsation could be felt in the dorsal artery of the left foot or in the posterior tibial artery. There was no evidence of cardio-vascular or other disease. An ulcer on the little toe had slowly healed, but cramplike muscular pains still occurred on walking. The disease had lasted about five years without the appearance of gangrene.

Weber calls this case one of arteritis obliterans with intermittent claudication.

CHAPTER VII.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS.

Arteriosclerosis is essentially a disease of middle life and old age; only rarely do we see it in persons under forty years of age.

The diagnosis of arteriosclerosis may be so easily made that the tyro could not fail to make it. It is, however, the purpose of this volume to lay stress on the earliest possible diagnosis and, if possible, to point out how the diagnosis may be arrived at. It is obviously much to the advantage of the patient to know that certain changes are beginning in his arteries, that, if allowed to go on, will inevitably lead to one or more of the symptom groups described in the preceding chapters.

The combination of (1) hypertrophied heart, (2) increased blood pressure, (3) palpable arteries, and (4) ringing, accentuated second sound at the aortic cartilage is in, reality, the picture of advanced arteriosclerosis. If the individual is in good condition much

may be done by judicious advice and treatment to ward off complications and prolong life with a considerable degree of comfort. But we should not wait until such signs are found before making a diagnosis and instituting treatment. As in all forms of chronic disease the early diagnosis is all important.

The history of the case is the first essential. Often a careful inquiry into the personal habits of a patient, with the record of all the preceding infectious diseases will give us valuable information and may be the means of directing the attention at once to the possible true condition. Particularly must we inquire into the family history of gout and rheumatism. An individual who comes of gouty stock is certainly more prone to arterial degeneration than one who can show a healthy heredity. Alcoholism in the family also is of importance because of the fact that the children of alcoholics start in life with a poor quality of tissue, and conditions that would not affect a man from healthy stock might cause early degeneration of arterial tissue in one of bad ancestry.

What infectious diseases has the patient had? Even the exanthemata may cause degenerations in the arteries, but, as has been shown, such lesions probably heal completely with no resulting damage to the vessel. Should the patient have passed through a long siege of typhoid fever the problem is quite different. Here, (vide supra) (Thayer) the palpable arteries do appear to be sclerosed permanently. Probably the length of time that the toxin has had a chance to act determines the permanent damage to the vessel wall. More potent than all other diseases to cause early arteriosclerosis is syphilis, and hence very careful inquiry should be made in regard to the possibility of infection with this virus. Not only the fact of actual infection but the duration and thoroughness of treatment are important matters for the physician to know.

What is the patient's occupation? Has he been an athlete, particularly an oarsman, has he been under any severe, prolonged, mental strain? Is he a laborer? If so, what form of manual labor is he engaged in? Such questions as these should never be overlooked as

they form the foundation stones of an accurate diagnosis and early, accurate diagnosis, we repeat, is essential to successful therapy.

We have called attention to the factor of sustained high pressure in the production of arteriosclerosis. Constant overstretching of the vessels leads to efforts of the body to increase the strength of the part or parts. The material which is used to strengthen the weakened walls has a higher elastic resistance than muscle and elastic tissue, but a lower limit of elasticity, and is none other than the familiar connective tissue. In athletes, laborers, brain workers who are under constant mental strain, and in those whose calling brings them into contact with such poisons as lead, there is every factor necessary for the production of high tension and consequently of arteriosclerosis.

Another question in regard to personal habits is how much tobacco does the patient use and in what form does he use it? Our experience is that the cigar smoker is more prone to present the symptoms of arteriosclerosis than the cigarette smoker, the pipe smo-

ker or the one who chews the tobacco. A very irritable heart results not infrequently from cigarette smoking but such is almost always found in young men in whom the lesions of arteriosclerosis are exceedingly rare. The probabilities are that the arteriosclerosis in cigar smoking results from the slowly acting poison which causes a rapid heart rate with an increase of pressure.

Last but not least, and perhaps the most important question is, has the patient been a heavy eater? This we believe to be a potent cause of splanchnic arteriosclerosis with the resulting indigestion, cramplike attacks, high blood pressure, etc. In a joking manner we are accustomed to remark "Overeating is the curse of the American people." There is however much truth in that sentence. Osler, than whom there is no keener observer, states that he is more and more impressed with the fact that overloading the stomach with rich or heavy or spiced foods is today one of the first causes of arterial degeneration. It stands to reason that this is true. We know that organs exposed constantly to hard work undergo hypertrophy, and that the blood

tension in those organs is high. Blood tension is, after all, dependent on capillary resistance, and if the capillaries are distended with blood, the resistance is great. The digestive organs can be no exception to this rule. Increased work means an increase of blood. This inevitably causes distension of the capillaries with stretching of the arteries and consequent damage to the walls. Once arteriosclerosis is present a vicious circle is established.

A man about forty-five consults us and says that he has noticed recently that he gets out of breath easily; in tying his shoes he experiences some dizziness. He finds that he has palpitation of the heart and possibly pain over the precordial region now and then. He notices also that he is irritable, that is his family tell him he is, and he notices that things that did not use to annoy him, now are almost hateful to him. On examination, one finds a palpable radial, a somewhat hypertrophied heart and slightly accentuated second aortic sound. The blood pressure may be high. The urine may or may not reveal any abnormalities. Not infrequently, although no albumen may be found, there are hyaline

casts. Such a case of arteriosclerosis is evidently not to be regarded as early. Then the question arises how are we to recognize early arteriosclerosis? I do not believe that the solution of this problem lies entirely in the hands of the physician. Some men are fortunate enough to come up for an examination for life insurance before an observant doctor who recognizes the palpable artery, makes out the beginning heart hypertrophy and the slightly accentuated second aortic sound. The patient will tell you that he never felt better in his life. He gets up at seven, works all day, plays golf, drinks his three to six whiskies, and is proud of his physical development. But the great mass of people are not fortunate from this standpoint. They do not seek the advice of the physician until they are stretched out in bed. They boast of the fact that for twenty years they have never had a doctor. One may well say that it is a problem how to reach such persons. It seems to me that there can be but one way to do this. The people must be taught that the duty of a physician is just as much to keep them in health as it is to bring

them back to health when they are ill. To that end people should be taught that at least twice a year they should be carefully examined. I do not mean that the patient should present himself to the doctor and, after a few questions the doctor say cheerfully "You are all right". The patient should be systematically examined. That means a removal of the clothing and examination on the bare skin. Such co-operation on the part of patient and doctor would save the patient years of active life and make of the doctor, what his position entitles him to, the benefactor to the community. Too often careless work on the physician's part lulls the patient into a false sense of security and he wakes up too late to find that he has wasted months or years of life. Early diagnosis of arteriosclerosis is only possible in exceptional cases unless people present themselves to the physician with the thought in mind that he is the guardian of health as well as the healer.

There are patients who go to the ophthalmologist for failing vision. Physically they feel quite well. They have been heavy eaters, hard workers, men and women who

have been under great mental strain. On examination of the fundus of the eye there is found slight tortuosity of the vessels with possibly areas of degeneration in the retina. A careful physical examination will usually reveal the signs of arteriocslerosis elsewhere. We have mentioned frequently high tension as an early sign. This must be taken with somewhat of a reservation, for this reason: not infrequently a persistent high tension is the earliest sign of chronic nephritis. The arteries may be pipe-stem in character and the heart small and flabby. However, if one watches for the palpably thickened superficial arteries (always bearing in mind the normal palpability as age advances) and the high tension, he cannot go far wrong in his treatment whether the case is one of chronic nephritis or of arteriosclerosis.

There is also this to bear in mind. Arteriosclerosis may be marked in some vessels and so slight in the peripheral vessels that it cannot with certainty be made out. But when the radials are sclerosed it is usually the case that similar changes exist in other parts. Then

too, there may be marked changes at the root of the aorta leading to sclerosis of the coronary vessels alone, and the first intimation that the patient or anyone else has that there is disease, may be an attack of angina pectoris. Except for symptoms on the part of the heart there is no way to make the diagnosis of sclerosis of the coronary arteries.

Differential diagnosis—In arriving at a diagnosis when the question is whether or not arteriosclerosis is the main etiological factor, the most important fact to know is the age of the patient. Other points that have been dwelt on fully must of necessity also be borne in mind.

Possibly the chief conditions that may be confused with some of the results of arteriosclerosis are pseudo agina pectoris which may be mistaken for true angina pectoris, and ulcer of the stomach, appendicitis (?) or other inflammatory abdominal condition which may mistaken for angina abdominalis.

Differential tables are sometimes of value in fixing the chief points of difference graphically.

Pseudo angina pectoris	True angina pectoris
Etiology rather certain; hysteria, neurasthenia, toxic agents, and reflex irritations.	Etiology not certain but almost always associated with arteriosclerosis of the coronary arteries and also aortic regurgitation.
No age is exempt. Usually in young people, chiefly females.	Age is important factor. Rare before forty, and males usually affected.
Paroxysms of pain occur spontaneously, are periodic and often nocturnal.	Paroxysms brought on by overexertions or excessive mental emotion. Rarely periodic.
Pain, while severe, is diffuse and sensation is of distension of heart. No sense of real anguish.	Intense pain, radiating down arm; heart felt as in a vise. Sense of anguish and impending dissolution.
Duration may be an hour or more.	Duration from few seconds to several minutes.
Restlessness and emotional symptoms of causative conditions are prominent.	Silent and fixed attitude, rigidity rather than restlessness.
Usually no increase in arterial tension.	Arterial tension is as a rule increased.
Prognosis favorable.	Prognosis most unfavorable.

In differentiating between ulcer of the stomach and angina abdominalis the following points may be of service:

Ulcer.	Angina abdominalis.
Occurs as a rule in young persons, more often females.	Only occurs in adults over forty who have been heavy eaters and drinkers, mostly males.
Pain of boring character increased by food and by certain positions with food in stomach. Felt through to left of spine.	Pain cramplike, diffuse, although more localized in epigastrium. Not necessarily any connection with food.
Occult blood found in stools.	No occult blood in stools
Considerable anemia apt to be present.	Anemia more often absent.
Arterial tension usually low.	Arterial tension high. (Splanchnic sclerosis)

DISEASES IN WHICH ARTERIOSCLEROSIS IS COMMONLY FOUND—There are certain more or less chronic diseases in which arteriosclerosis is found either as a separate disease or as a result of the chronic disease itself, or the sclerosis may be the cause of the disease. As examples of the first class are diabetes mellitus, and cirrhosis of the liver. As examples of the second class are chronic nephritis, gout, syphilis, lead poisoning. Examples of the third class have already been fully described. Then certain rare diseases

that have been briefly described in this chapter, viz: Raynaud's disease and erythromelalgia, are frequently associated with demonstrable arteriosclerosis.

CHAPTER VIII.

PROGNOSIS.

In a disease that presents as many vagaries as arteriosclerosis, it is not possible to give a certain prognosis. Unfortunately we do not as a rule see the arteriosclerotic until the disease is well advanced, or even after some of the more serious complications have taken place. By that time the condition is progressive, and while the prognosis is grave the individual may live a number of years.

It is fortunate for the arteriosclerotic that mild grades of the disease are compatible with a fairly active life. The disease in this stage may become arrested and the patient may live many years. Not only in the mild grades is this possible. Even patients with advanced sclerosis may enjoy good health provided the organs have not been so damaged as to render them unfit to perform their functions. The frequency with which we see advanced arteriosclerosis at the post mortem table as an accidental discovery, attests the truth of the foregoing statement. Yet how often

does it happen that individuals, apparently in the best of health, suddenly succumb to an asthmatic or uremic attack, an apoplexy, cessation of the heart beat or a rupture of the heart due to arteriosclerosis!

In order to arrive at an intelligent opinion in regard to prognosis certain factors must be taken into consideration, chief of which are; the seat of the sclerosis, the probable stage, the existing complications, and, last and most important, the patient himself. The whole man must be studied and even then our prognosis must be most guarded.

It is much more dangerous for the patient when the process is in the ascending portion of the arch of the aorta than when it has attacked the peripheral arteries. Here, at the root of the aorta, are the openings of the coronary arteries and the arteries supplying the brain are close by. The coronary arteries here control the situation. When loud murmurs are heard at the aortic orifice and the heart is evidently diseased, it is useful to divide the endocarditis into two types, the arteriosclerotic and the endocarditic. The etiology of the former is sclerosis and the

prognosis is grave because of the liability, nay the probability, that the orifices of the coronary arteries will become narrowed. The etiology of the second type is in most cases rheumatic fever or some other infectious disease, and the prognosis is far better than in the first type. True, the two may be combined. In such a case, the prognosis is entirely dependent upon the course of the arteriosclerosis.

The involvement of the arteries in the kidneys is of considerable importance for it is usually bilateral and widespread. As a rule the disease makes but slow progress provided that the general condition of the patient is good, but at any time from a slight indiscretion or for no assignable cause, symptoms of renal insufficiency may appear and may rapidly prove fatal.

It must not be thought that because the localisation of the arteriosclerosis in the peripheral arteries is usually the most favorable condition that it is therefore devoid of ill effects. On the contrary, very serious, even fatal, results may be brought about by interference with the circulation with resultant extensive gangrene

of the part supplied by the diseased arteries. The amputation of a portion of a leg, for instance, may relieve, to some extent, an overburdened heart and prove life-saving to the patient, but the neuritic pains are not necessarily relieved. The torture from these pains may be excruciating.

No stage of the disease is exempt from its particular danger. In the early stages of the disease before the artery or arteries have had time to become strengthened by proliferation of the connective tissue, there is the danger of aneurysm. Later, the very same protective mechanism leads to stiffening and narrowing of the arteries and hence to increased work on the part of the heart with all of its consequences. Thrombosis is favored, and where atheromatous ulcers are formed, embolism is to be feared.

As the complications and results of arteriosclerosis come to the front every one must be considered by itself and as if it were the true disease. There may be a slight apoplectic attack from which the patient fully recovers, but the prognosis is now of a grave character as the chances are that another

attack may supervene and carry off the subject. Yet after an apoplectic attack patients have lived for many years. Probably the most noted illustration of this is the life of Pasteur. He had at forty-six hemiplegia with gradual onset. He recovered with a resulting slight limp, did some of his best work after the stroke, and lived to be seventy-three years old. Yet the exception but proves the rule and the prognosis after one apoplectic stroke should always be guarded.

The first attack of cardiac asthma is to be looked upon as the beginning of the end. The end may be postponed for some time but it comes nearer with every subsequent attack. One may recover from what appears to be a fatal attack of cardiac asthma accompanied by oedema of the lungs and irregular, intermittent, laboring heart, but the recovery is slow and the chances that the next attack will be the fatal one are increased.

The significance of albuminuria is difficult to determine. The kidneys secrete albumen under so many conditions that the mere presence of albumen in the urine may have but lit-

tle prognostic value. Many cases are seen where there is no demonstrable albumen, and yet the patient may suddenly have a cerebral hemorrhage. As a general rule the urine should be carefully examined but not too much stress should be laid on the discovery of albumen and casts. It is not always possible to determine the extent of the kidney lesion by the urinary examination, yet at any time a uremic attack may appear and prove fatal. One might say that the appearance of albumen in the urine of an arteriosclerotic where it had not been before, is a bad sign, and in making a prognosis this must be taken into consideration.

Bleeding from the nose is not infrequently seen in those who have arteriosclerosis. It can hardly be called a dangerous symptom as it can always be controlled by tampons. There are times when epistaxis is decidedly beneficial as it relieves headache, dizziness, and may avert the danger of a hemorrhage into the brain substance. It is rare to have nose bleed except in cases of bigh tension in plethoric individuals. My experience has been that it has saved me the trouble of

bleeding the patient. It is always of serious import in that it indicates a high degree of tension, but there is scarcely ever any immediate danger from the nose bleed itself.

Intestinal hemorrhage is always a grave sign. As has been shown, arteriosclerosis of the splanchnic vessels not infrequently occurs, and an embolus or thrombus may completely occlude the superior mesenteric artery. The chances of the establishment of a collateral circulation are small, as the arteries of the intestines are end arteries. Necrosis of the part follows, blood is found in the stools, and perforation or gangrene, or both, are apt to follow. There may be blocking of small branches only leading to ulceration of the intestine. Under all conditions the prognosis is serious.

The general condition of the patient, his build, physical strength, powers of recuperation, etc., must be taken into account in giving a prognosis. The more powerful the individual the more favorable as a rule is the prognosis with this reservation always in mind, that the greater the body development the greater is the heart hypertrophy and the

accidents from high tension must not be overlooked. Many puny individuals with stiff, calcified arteries go about with more ease than a robust man with thickened arteries only. The differentiation as pointed out by Allbutt, is well to keep in mind in giving a prognosis. It cannot be too strongly emphasized that it is the whole patient that we must consider and not any one system, that at the time happens to be the seat of greatest trouble, and by its group of symptoms dominates the picture.

It is evident from what has been said that an accurate prognosis in arteriosclerosis is no easy matter. Were arteriosclerosis a simple disease of an acute character there might be grounds for giving a more or less definite prognosis. The most that can be said is that arteriosclerosis is always a serious disease from the time that symptoms begin to make themselves known. The gravity depends altogether on the seat of the greatest arterial changes, and is necessarily greater when the seat is in the brain than when it is in the legs or arms.

The attitude of the patient himself also determines to a great extent the prognosis. Some men, especially those who have always enjoyed good health, turn a deaf ear to warnings and instead of ordering their lives according to the advice of the physician, persist in going their own way in the hope that the luck that has always been with them will continue to stand at their elbows. Neither firmness nor pleadings avail with some men. The only salve for the conscience of the physician is that he has done his best to steer the patient away from the shoals and breakers. In others who realize their condition and take advantage of the advice given as to the regulation of their lives, the prognosis is generally favorable.

To sum up the chapter in a few words, we should say: Always remember that the patient is a human being, study his habits and character and mode of life; look at him as a whole; take everything into consideration, and give always a guarded prognosis.

CHAPTER IX.

PROPHYLAXIS.

Arteriosclerosis comes to almost everyone who lives out his allotted time of life. As has been noted within, many diseases and many habits of life are conducive to the early appearance of arterial degeneration. Decay and degeneration of the tissues are necessary concomitants of advancing years and none of us can escape growing old. From the period of adolescence certain of the tissues are commencing a retrograde metamorphosis, and hand in hand with this goes the deposit of fibrous tissue which later may become calcified. The arterial tissue is no exception to this rule, and we have already shown that certain changes normally take place as the individual grows older, changes which are arteriosclerotic in type and are quite like those caused in younger people by many of the etiological factors of the disease.[1]

We are absolutely dependent upon the integrity of our hearts and blood vessels. Respiration may cease and be carried on ar-

tificially for many hours while the heart continues to beat. Even the heart has been massaged and the individual has been brought back to life after its pulsations have ceased, but such cases are few in number. We cannot live without the heartbeat and the prophylaxis of arteriosclerosis consists in the adjustment of our lives to our environment, so that we may get the maximum amount of work accomplished with the minimum amount of wear and tear on the blood vessels.

The struggle for existence is keen. Competition in every profession or trade is exceedingly acute, so much so that to rise to the head in any branch of human activity requires exceptional powers of mind. Among those who are entered in this keen competition, the fittest only can survive for any period of time. The weaklings are bound to succumb. A scion of halthye stock will stand the wear and tear far better than will the progeny of diseased parentage.

It is only necessary to call attention to the part that alcohol, syphilis and insanity play in heredity. These have been discussed fully in the earlier part of this book.

We live rapidly, burning the candle at both ends. It is not strange that so many comparatively young men and women grow old prematurely. While heredity is a factor as far as the prophylaxis of arteriosclerosis is concerned, of far more importance is the mode of life of the individual. Scarcely any of us lead strictly temperate lives. If we do not abuse our bodies by excessive eating and drinking and so wear out our splanchnic vessels and cause general sclerosis by the high tension thereby induced, we abuse our bodies by excessive brain work and worry with all their multitudinous evils. The prophylaxis of arteriosclerosis might well be labeled "The Plea for A More Rational Mode of Life". Moderation in all things is the keynote to health, and to grow old gracefully is an art that admits of cultivation. Excesses of any kind be they mental, moral, or physical, tend to wear out the organism.

People habitually eat too much; many drink too much. They throw into the vascular system excessive fluid combined with toxic products that cause eventually a condition of high arterial tension. It has been

shown how poisonous substances absorbed from the intestines have some influence on the blood pressure. Anything that causes constant increase of pressure should be studiously avoided.

Mild exercise is an essential feature of prophylaxis. One may, by judicious exercise and diet, make of himself a powerful muscular man without, at the same time, raising his average blood pressure. The man who goes to excess and continually overburdens his heart, will suffer the consequences, for the bill with compound interest will be charged against him. It is a great mistake for anyone to work incessantly with no physical relaxation of any kind, and yet, after all, it is not so much physical relaxation that is necessary, as the pursuit of something entirely different, so that the mind may be carried into channels other than the accustomed routes. Diversification of interests is as a rule restful. That is what every man who reaches adult life should aim at. Hobbies are sometimes the salvation of men. They may be ridden hard, but even then they are helpful in bearing one completely away from daily cares and worries.

The man who can keep the balance between his mental and physical work is the man who will, other things being equal, live the longest and enjoy the best health.

Nowadays the trend of medicine is towards prophylaxis. We give the State authority to control epidemics so far as it is possible by modern measures to control them.

We urge over and over again the value of early diagnosis in all chronic diseases, for we know that many of them, and this applies particularly to arteriosclerosis, could be prevented from advancing by the recognition of the condition and the institution of proper hygienic and medicinal treatment.

It is the patent duty of every physician to instruct the members of his clientele in the fundamental rules of health. Recently the President of the American Medical Association, in his address before the 1908 meeting, urged the dissemination of accurate knowledge concerning diseases among the laity. While this may be done by City and State Boards of Health, it seems far better for the modern trained physician to work among his own people. With concise information

concerning the modes of infection and the dangers of waiting until a disease has a firm hold before consulting the health mender, people should be able to protect themselves from infections and be able to nip chronic processes in the bud. But it is difficult to turn the average individual away from the habit of having a drug-clerk prescribe a dose of medicine for the ailment that troubles him. It is really unfortunate that most of the pains and aches and morbid sensations that one has speedily pass away with little or no treatment. Herein lies the strength of charlatanism and quackery. Unfortunate, yes, for a man can not tell whether the trivial complaint from which he suffers is any different from the one that was so easily conquered six months ago. But instead of recovering, he grows worse. Hope that springs eternal in the human breast, leads him to dilly-dally until he at last seeks medical advice, only to find that the disease has made such progress that little can be done.

Instruct the public to consult the doctors twice a year. The dentists have their patients return to them at stated intervals

only to see if all is well. How much more rational it would be if men and women past the age of forty had a physical examination made twice a year to find out if all is well.

The prophylaxis of arteriosclerosis is moderation in all the duties and pleasures of life. This in no sense means that a man has to nurse himself into neurasthenia for fear that something will happen to him. As one grows in years exercise should not be as violent as it was when younger, and food should be taken in smaller quantities. Many forms of exercise suggest themselves, particularly walking and golf. Walking is a much neglected form of exercise which, in these modern days with our thousand and one means of locomotion, is becoming almost extinct. There is no better form of exercise than graded walking. To strengthen the heart selected hill climbing is one of the best therapeutic methods that we have. The patient is made to exercise his heart just as he is made to exercise his legs, and as with exercise of voluntary muscles comes increase in strength, so by fitting exercise may the heart muscle be increased in power. A warning

should be sounded however against over exercise. This leads naturally to hypertrophy with all its disastrous possibilities. Men who have been athletes when young should guard against overeating and lack of exercise as they grow older. Many of the factors which favor the developement of arteriosclerosis are already there and a sedentary, ordinary life such as office all day, club in afternoon, a few drinks and much rich food, will inevitably lead to well advanced arterial disease.

Karl Marx in his famous Socialistic platform said "No rights without duties; no duties without rights." So we may paraphrase this and say "No brain work without moderate physical exercise in the open air; no physical exercise without moderate brain work."

There is yet one other point that is important, the combination of concentrated brain work and constant whiskey drinking. This is most often seen in men of forty-five to fifty-five, heads of large business concerns who habitually take from six to twelve drinks of whiskey daily, and with possibly a bottle of wine for dinner. Such men look ruddy and in

prime health but almost invariably, careful examination will reveal unmistakable signs of arterial disease. There is usually the enlarged heart and pulse of high tension with or without the trace of albumen in the urine. The lurking danger of this group of manifestations has so impressed the medical directors of several of the large insurance companies that a blood pressure reading must be made on all applicants over forty years of age. Should high blood pressure be found the premium is increased as the expectation of life is proportionately shorter in such men than in normal persons.

Therefore, let every physician act his part as guardian of health. Only in this way is the prophylaxis of arteriosclerosis possible.

CHAPTER X.

TREATMENT.

Although it has been rather dogmatically stated (vide supra) that everyone who reaches old age has arteriosclerosis, it must not be inferred that absolutely no exceptions to this rule are found. Cases are known where persons of ninety years even had soft arteries, and we have seen persons of sixty whose arteries could not be palpated. When infants and children are seen with considerable sclerosis, it proves that, after all, it is the quality of the tissue even more than the wear and tear, that is the determining factor in the production of arteriosclerosis. It would be well if those who cannot bring healthy progeny into the world were to leave this duty to those who can.

In general the treatment of arteriosclerosis is prophylactic and symptomatic. In the preceding chapter we had something to say about prophylaxis in general; we must again refer to it in detail.

Arteriosclerosis is essentially a chronic progressive disease, and the secret of success in the management of it is not to treat the disease or the stage of the disease, but to treat the patient who has the disease. To infer the stage of the disease from the feeling of the sclerosed artery, may lead to serious mistakes. Persons with calcified arteries may be perfectly comfortable, while those with only moderate thickening may have many severe symptoms. The key-note is individualisation. It is manifestly absurd to treat the laboring man with his arteriosclerosis as one would treat the successful financier. The habits, mode of life, every detail, should be studied in every patient if we expect to gain the greatest measure of success in the treatment. One may treat fifty patients who have typhoid fever by a routine method and all may recover. Individualising, while of great value in the treatment of acute diseases, yet is not absolutely essential in order that good results may be obtained. Far different is it when treating a disease like arteriosclerosis. One who relies on textbook knowledge will find

himself at a loss to know what to do. Text-books can only outline, in the briefest manner, the average case, and no one ever sees the average book case. At the bedside with the patients is the place to learn therapeutics as well as diagnosis. All that can be hoped for in outlining the treatment of arteriosclerosis is to lay down a few principles. The tact, the intuition, the subtle something that makes the successful therapeutist, can not be learned from books. So the man who treats cases by rule of thumb is a failure from the beginning. There are certain general principles that will be our sheet anchors at all times and for all cases. The art of varying the application of these fundamentals to suit the individual case, is not to be culled from printed words.

Hygienic treatment—Every man is more or less the arbiter of his own fate. Granted that he has good tissue to begin life, his own habits and actions determine his span of comfortable existence. No one cares to live after his brain begins to fail and the failing brain is generally due to disease of the cranial arte-

ries. The hygienic treatment resolves itself into advice in regard to prophylaxis.

First and foremost is exercise. It has seemed to us that the revival of out-of-door sports is one of the best signs of promise of the preservation of a virile, hardy race. That women, as well as men, indulge in the lighter forms of out-of-door exercise should bring it about that the coming generation all start in life under the most advantageous conditions of bodily resistance.

Among all the forms of exercise, golf probably is the best. It is not too violent for the middle aged man, yet it gives the young athlete quite enough exercise to tire him. It is played in the open. One is compelled to walk up and down in pleasant company, for golf is essentially a companionable game, while he reaps the full benefit of the invigorating exercise. The blood courses through the muscles and lungs more rapidly: the contraction of the skeletal muscles serves to compress the veins and so to aid the return of blood to the heart: the lungs are rendered hyperemic, deeper and fuller breaths must be taken; oxidation is

necessarily more rapid, and effete products, which if not completely oxidized would possibly act as vasoconstrictors, are oxidized to harmless products and eliminated without irritating the excretory organs.

Other forms of out-of-door exercise that can be recommended are tennis, canoeing, rowing, fishing, horseback riding, swimming, etc. Tennis is the most violent of all the sports mentioned and might readily be overdone. Rowing as practised by the eights at college is undoubtedly too violent a form of exercise, and may be productive in later life of very grave results. Canoeing is a delightful and invigorating exercise. The muscles of the arms, shoulders, and trunk are especially used, the leg muscles scarcely at all. Nevertheless the deep breathing that necessarily comes with all chest exercises aerates every portion of the lungs, and is of great benefit to the whole body.

Swimming as an exercise has much to recommend it. In this sport all the muscles take part and at the same time the chest is broadened and deepened.

All these methods of using the muscles to keep ourself in trim, so to speak, are part and parcel of the general hygienic mode of life that is conducive to a healthy old age. Exercise can be overdone, as eating can be overdone. Both are essential and yet both can be the means of hastening an individual to a premature grave.

When the arteriosclerosis has advanced so far that it is easily recognizable, certain forms of exercise should be absolutely prohibited. Such are tennis, rowing, swimming. Horseback riding to be allowed must be strictly supervised. At times this may be an exceedingly violent exercise. As an out-of-door sport, there is nothing that equals golf. The physician, knowing the character of the course, and the length of it, can say to his patient that he may play six, nine, twelve, or eighteen holes, depending on the patient's condition.

For those who are not able to get out, exercise in the room with the windows open must take the place of out-of-door sports. Here the use of chest weights is a most excellent means of keeping up to the tone of the

muscles. By adjusting the weights, the exercise may be made light, medium, or heavy. Every physician should be familiar with the chestweight exercises. They are not as good as open air exercise but they undoubtedly have been the means of saving years of life to many patients with arterial disease.

There comes a time when all forms of exercise must be prohibited on account of the dyspnoea, oedema, dizziness, etc. It seems unwise to keep such a patient in bed, even though the oedema be considerable. Once on his back in bed he becomes weak, and the danger of oedema of the lungs or hypostatic congestion of the bases, with subsequent broncho-pneumonia, is very great. Although such persons can not exercise actively, they should have passive exercise in the form of massage, carefully given, so that no injury is done to the rigid vessels. It is possible to rupture a vessel, the walls of which are encrusted with lime salts, and full of small aneurysmal dilatations. Every patient must be watched carefully and measures instituted for the individual.

BALNEOTHERAPHY—As a bracer and invigorator, the cold or cool bath, (shower or tub) in the morning on arising can be highly recommended. It promotes skin activity, is a stimulant to the bowels and kidneys and to the general circulation, besides being cleansing. We find today that the morning bath has become such a necessity to the average American that all new hotels are fitted with private baths, and old hotels, in order to get patronage, are arranging as many baths connected with sleeping rooms as is possible. Our generation assuredly is a ruddy, clean-bodied one. What the actual results of this out-door life and frequent bathing will be for the race remains to be seen, but one cannot but feel that it must build up a stronger, more resistant race of people, who not only enjoy better health than did their forefathers, but enjoy it longer.

Not every one can stand a cold bath. It is folly to urge it on one to whom it is distasteful, or on one who does not feel the comfortable glow that should naturally result. For the well, or those with a tendency to arteriosclerosis, or those in whose families there

have been several members who had early arteriosclerosis, such proceedings as recommended could not be improved upon. However, for the person who has well recognized sclerosis, only warm baths should be advised, and these not daily. The water should be at a temperature of 90–95 degrees F. Care should be taken that persons sent to spas be cautioned against hot baths. It is not inconceivable that the increased force of the heart beat that accompanies a hot bath might be sufficient to rupture a small cranial vessel. Hence, Turkish and Russian baths should be most unqualifiedly condemned. As a matter of fact, persons vary so in their habits with regard to bathing that what might suit one person would do another much harm.

PERSONAL HABITS—The personal habits of the individual, more than any other factor, determine whether or not arteriosclerosis sets in early in his life. The man or woman who is moderate in eating and drinking, sees that the kidneys are kept in good condition, and attends strictly to regularity of the bowels, lays a good basis for the measure of health which is so essential for happiness. It has been

shown that sclerosis of the splanchnic vessels may be due to constant irritation of toxic products elaborated in digesting constantly enormous meals. In obstinate constipation, many poisons, the nature of which we do not know, are absorbed and circulate in the blood. We have not sufficient data to prove that constipation favors the production of arteriosclerosis, but our impression has been that it does favor it. Constipation can often be relieved by a glass of water before breakfast, a regular time to go to stool, and abdominal massage or exercises. Some maintain that it is a bad habit only, and can be readily overcome. Whatever is done, avoid leading the patient into the drug habit, for the last state of the patient will be worse than the first. Habits of sleep are not of such great importance. Most persons get enough sleep except when under severe mental strain. Most adults need from seven to eight hours sleep, although some can do all their work and keep in prime health on five or six hours sleep.

Tobacco has been accused of causing many ills and has been thereby much maligned.

We can not see that the use of tobacco in any form in moderation is harmful to most men. Undoubtedly the blood pressure is raised when mild tobacco poisoning occurs, and individual peculiarities of reaction to the weed are multitudinous. But to condemn offhand the use of plant is the height of folly. There is no reason why the arteriosclerotic who has always used tobacco in moderation, should not continue to use it, whether he smoke cigarettes, cigars, or pipe. His supply should be decreased, but there is no sense in depriving a man of one of the solaces of life, unless, as is sometimes the case, abstinence is easier to the patient than moderation.

As for alcohol, opinions differ widely. Some see in alcohol one of the most frequent causes of arteriosclerosis; others do not believe that the part played by alcohol is a serious one; only in conjunction with other poisonous substances is it dangerous. Probably unreasoning fanaticism has had much to do with the wholesale condemnation of alcoholic beverages. The general effect of alcohol is to lower the blood pressure by causing marked dilatation of all the vessels of the skin. True,

the alcohol circulates in the blood, and is broken up in the liver, and this organ would seem to bear the brunt of the harm done. Alcoholic drinks in moderation, I do not believe have any deleterious effect on health. On the contrary, I believe that they may in some cases assist digestion and assimilation. Indiscriminate indulgence is to be condemned, as is over indulgence in exercise or eating. What may be moderate for A, might be excessive for B. Every man is then the arbiter of his own fortune and within his own limits can indulge moderately (a relative term after all) without fear of doing himself harm. In advanced arteriosclerosis it is necessary to decrease the supply of alcohol just as it is necessary to cut down the food supply. This must rest entirely on the judgment of the physician, who must not act arbitrarily, but must have his reasons for every one of his orders.

DIETETIC TREATMENT—Most persons eat too much. We not only satisfy our hunger, but we satisfy our palates, and, instead of putting substantial foodstuffs into our stomachs, we frequently take unto ourselves concoctions that defy description.

Food stuffs are composed of one or all of three clases: (1) proteids, (2) fats, (3) carbohydrates. As examples of the first are beef and white of egg; of the second, the oils, butter, lard; of the third, sugar, potato, beet, corn, etc.

The physiologists and chemists have shown us that both endogenous and exogenous uric acid in excess will cause a rise of blood pressure, but the bodies most concerned in the production of elevated blood pressure are the purin bodies, those organic compounds which are formed from proteids, and represent chemically a step in the oxidation of part of the proteid molecule to uric acid. Red meat contains more of the substances producing purin bodies than any other one common food stuff, and for this reason the excessive meat eater is, *ceteris paribus*, more apt to develop arteriosclerosis comparatively early in life. An amusing experience of Dr. J. Mackenzie's is *apropos*. He writes, "An elderly man came to see me complaining of slight attacks of angina pectoris. His arteries were thickened and his pulse very hard, 210 mm. Hg. He is a brewer's agent.

I said, 'You must give up beer and spirits.' He replied, 'I am a teetotaler.' 'Well, then, you must eat less butcher's meat'. 'I'm a vegetarian' was his reply!''

Nevertheless, for the majority of persons too much meat is undoubtedly harmful.

The fats and carbohydrates contain practically no substances that react on the body of the ordinary individual in a deleterious manner during their digestion. The extra work that is put on the heart by the formation of many new blood vessels in adipose tissue is the only harmful effect of over indulgence in these food stuffs.

It has been found that nitrogen equilibrium can be maintained at a wide range of levels. Formerly 135–150 gms. of proteid daily were considered necessary for a man doing light work. Now it is known that half that amount is sufficient to keep one in nitrogenous equilibrium, and to enable one to keep his weight. A person at rest requires even less than that. One who is engaged in hard physical labor burns up more fuel in the muscles, and so must have a larger fuel supply.

Although we habitually eat too much we drink too little water. For those who have any form of arterial disease an excess of fluid is harmful, as the vessels become filled up and a condition of plethora results, which necessarily reacts injuriously on the heart and circulation. The drinking of a glass of water during meals is, I believe, good practice. The water must be taken mouthful at a time, and not gulped down. If this is done, there results sufficient dilution of the solid food to enable the gastric juices successfully and rapidly to reach all parts of the meal.

Some are in favor of a rigid milk diet for those who have arteriosclerosis. Some men have lived on nothing but milk for several years and have not only kept in good health, but have actually gained weight and led at at the same time active lives. It has been held by others that rigid milk diet is positively harmful on account of the relatively large quantity of calcium salts that are ingested. This was thought to favor the deposition of calcareous material in the walls of the already diseased arteries. While possibly there may

be some danger of increased calcification, the majority of clinicians are in favor of a milk cure given at intervals. Thus the patient is made to take three to four quarts daily for a period of a month. There is then a gradual return to a general diet, exclusive of meat, for several weeks, then another rigid milk diet period.

If we are bold enough to follow Metchnikoff in his theories of longevity, we might advise resection of the large intestine, on the ground that it is an enormous culture tube that produces prodigious amounts of poisonous substances which are thrown into the general circulation. To combat such a grave (?) condition as the carrying of several feet of large intestine, we are recommended to take buttermilk or milk soured by means of the *b. acidus lacticus*. Clinical experience has taught that in arteriosclerosis buttermilk is of great value, whether it be the natural product, or made directly from sweet milk by the addition of the bacilli. The latter is a smoother product and has, to my mind, a delightful flavor. Cases that cannot take milk or any other food will often take butter-

milk, and do well on this restricted diet. From two to four quarts daily should be taken. It should be drunk slowly as should milk.

MEDICINAL—It has long been thought that the iodides have some specific effect on the advancing arteriosclerosis, checking its spread, if not really aiding nature to a limited restoration of the diseased arteries. It is possible that the eulogies upon the iodides owe their origin to the successful treatment of syphilitic arteriosclerosis, in which condition these drugs have a specific action. However that may be, there is no doubt that the administration of sodium or potassium iodide is good therapeutics in cases of arteriosclerosis.

Unfortunately many persons have such irritable stomachs that they cannot take the iodides, even though they be diluted many times. They may be made less irritating by giving them with essence of pepsin. Unless the case is syphilitic, it is doubtful if it is of value to increase the dose gradually until a dram or even more is taken three times daily after meals. Usually a maximum dose of ten grains seems to be quite sufficient. This

may be taken three times a day, well diluted, for three months. There follows a month's rest, then the treatment is resumed for another period of three months, and so on. Either sodium or potassium iodide in saturated solution may be given. The sodium salt is possibly less irritating, and contains more free iodine than the potassium salt, although the latter is more generally used. The strontium iodide may also be used.

One sees a patient now and then who cannot take the iodides, however they may be combined. For such patients one may obtain good results with iodopin, sajodin, or other of the preparations put up by reputable firms. Personally, I have never yet seen a patient who could not take the ordinary iodides in some form or other, and I am opposed to ready made drugging.

The action of the iodides is to lower the blood pressure, and they are of greatest value when the blood pressure is high, and when headache, and precordial pain are present.

When the case is moderately advanced, very mild doses, gr. $\frac{1}{3}$ morning and evening, of the thyroid extract may be given. It is

generally believed that the internal secretion of the thyroid and the adrenal are antagonistic. That the thyroid secretion lowers blood pressure is certain, possibly on account of its iodine content. Some combinations of iodine and thyroid such as the iodothyroidin have been used and have had some measure of success attributed to them.

When the blood pressure is high and there is reason to believe that this should be controlled, we have at hand a group of drugs which have proved of inestimable value. The nitrites have the power of markedly reducing the pressure, and of equalizing the circulation. The most evanescent of these drugs is amyl nitrite. This is put up in the form of capsules, or pearls, containing from one to three minims, which, when needed, are broken in a cloth under the nostrils. The effect is almost instantaneous. There is flushing of the face and other peripheral vessels denoting a relaxation and widening of the bed of the blood stream, and a consequent decrease in the pressure in the arteries. The effects of amyl nitrite however are soon over. It is used only in attacks of cardiac spasm, as in

angina pectoris. Nitroglycerin, the spiritus glonoini of the U. S. P., acts in about the same manner as amyl nitrite but the effects last longer. One drop of the one per cent. solution given every four hours and increased to physiological effect, then reduced just below the dosage at which disagreeable effects follow, is a very valuable means of reducing pronounced high tension. I have found this drug of great benefit especially in cases where arteriosclerosis combined with chronic nephritis causes cardiac asthma. Still another drug which I have found of service in these conditions, one whose sphere of action is somewhat broader, because its effects are more lasting, is sodium nitrite. This is given in water in doses of one to three grains every four hours. Some have objected to the use of this drug, but my experience has made me place much confidence in its harmlessness, provided that the patient is carefully watched. This, however, applies to all of the nitrite compounds. For a mild case, one often finds that sweet spirits of nitre is sufficient to control the pressure and relieve the distressing symptoms, and it is undoubtedly the least

harmful of all the nitrites. Drugs that are of great value, but of which little is noted in textbooks, are aconite and veratrum viride. Both of these drugs are well known to be marked circulatory depressors. Veratrum viride in my experience should be very cautiously used, and never used unless a trained attendant is constantly at hand. With regard to aconite I have no such feeling, and a mixture of tincture of aconite and spiritus aetheris nitrosi may be given for several weeks with no fear of doing any harm. Personally, of all the drugs mentioned, I prefer the nitrite of sodium or the combination just given. They may be advantageously alternated.

After all, as a pressure reducer, no drug or group of drugs can take the place of absolute rest in bed with careful regulation of the hygiene and diet of the patient. This should be borne in mind and a course of this treatment should be instituted in all cases of persistent high tension in which symptoms are present.

With the fibrolysin of Merck, I have had no experience. Some men assert that they

have had good results from its use, but on the whole the evidence is not highly favorable.

Morphine is invaluable. No drug is of such value in the nocturnal dyspnoeic attacks that occur in the late stages of arteriosclerosis when the heart or the kidneys are failing. Morphine not only relaxes spasm and quiets the cerebral centres, but is an actual heart stimulant under such conditions, and should never be withheld, as the danger of the patient's becoming addicted to its use is more fanciful than real.

As heart stimulants, one may use strychnine, spartein, caffein, or camphor. In desperate cases, where a rapidly diffusible stimulant is needed, a hypodermic syringefull of ether may be given, and repeated in a short while.

Several years ago a so-called serum was brought out by Trunecek which was said to have a favorable effect on the metabolism of the vessel walls. It was given at first hypodermatically or intravenously but the former method was painful. It was later stated that given by mouth it acted just as well. The results with the Trunecek serum have not come up

to the expectations that the early favorable reports promised. The original serum was composed as follows: NaCl, 4.92 gm.: $Na_2 SO_4$, 0.44 gm.; $Na_2 CO_3$, 0.21 gm.: $K_2 SO_4$, 0.40 gm.; aqua destil. q. s. ad. 100.0 cc. Later this was modified for internal use to the following prescription·

R

Natrii chlor.	10. gm.
Natrii sulphat.	1. gm.
Natrii carbonat.	0.40 gm.
Natrii phosphat.	0.30 gm.
Calcii phosphat.	
Magnesii phosphat.	aa 0.75 gm.

M. Ft. cachets No. XIII.

The contents of every cachet corresponds to 15 cc. of the fluid serum or to 150 cc. of blood serum. The preparation called antisclerosin consists of the salts contained in the serum. As to its efficacy, I can not judge, as I have never felt that it was worth while to use it. Reports of cases in which it has been tried do not speak very highly of it.

In the general treatment of arteriosclerosis, there is no one factor of more importance than the regular daily bowel movement.

Attention to this may save the patient much discomfort and even acute attacks of cardiac embarrassment. The choice of the purgative is immaterial, with this reservation only, that the mild ones, such as cascara, rhubarb, licorice powder and the mineral waters, should be thoroughly tried before we resort to the more drastic purgatives. The old Lady Webster dinner pill is an excellent tonic aperient. When the heart is embarrassed and oedema of the legs and effusion into the serous cavities have taken place, then it becomes necessary to use the drastic purgatives that cause a number of watery movements. Epsom salts given in concentrated form, elaterin gr. 1-12, the compound cathartic pill, blue mass and scammony, or even croton oil may be used. Since the observation of a greatly congested intenstine from a patient who had been given croton oil, I have ceased to use this purgative, and I doubt much if its use is ever justifiable in these cases.

The management of the ordinary case of arteriosclerosis resolves itself into a careful hygienic and dietetic regime with the addition of the iodides, aconite, or the nitrites.

A diet consisting of very little meat, alcohol in moderation or even absolutely prohibited, and not too much fluid should be prescribed. Condiments and spices should also be used sparingly. Cold baths, shower baths, cold and hot sheets alternating, are of great benefit in assisting the heart to do its best work by making the large capillary area of the skin more permeable. It is not true that such baths raise the blood pressure so markedly. Certain acts,' as sneezing, violent coughing, etc., increase the blood pressure much more than judicious bathing.

SYMPTOMATIC TREATMENT—The fact that arteriosclerosis really loses much of its own identity and, in later stages, becomes merged with the symptomatology of the diseases of various organs, as the kidney, brain, heart, compels us for completeness' sake to say a few words about the treatment of these complications.

One of the results of arteriosclerosis of the coronary arteries, angina pectoris, demands prompt treatment. In the acute attack, the chief object is to relieve the spasm and pain. Pearls of amyl nitrite should be inhaled,

and morphine sulphate with atropine sulphate given hypodermatically at the very earliest moment. It is senseless to withhold morphine. The only possible reason for withholding it would be uncertainty as to the diagnosis. It is probably better to err on the safe side, and should the case prove to be one of pseudo angina, in the next attack sterile water can be given instead of the morphine and atropine.

When a patient is seen in the condition of broken compensation with the much dilated heart, anasarca, dyspnoea and suppression of urine, there is no better practice than venesection. Especially is this valuable when the tension is still fairly high and the individual is robust. Following the abstraction of six to eight ounces of blood the whole picture changes, so that a man who a short while before was apparently at death's door, notices his surroundings and takes an interest again in life. This should be followed up with thorough purgation, and cardiac stimulants should be ordered. In such cases digitalis is useful, but its action is never so striking as in cases of this general character

due to uncompensated valvular disease. It must be remembered that in arteriosclerosis the changes in the myocardium must be of a considerable grade for the heart to give away. Therefore, digitalis can not be expected to act on a diseased muscle as it acts on a comparatively healthy muscle. It is only in such cases of broken compensation that digitalis should ever be used. It is a vasoconstrictor as well as a cardiac stimulant, and hence in choosing a drug to increase the working power of the heart when there is only arteriosclerosis and a weak heart, one should put digitalis out of the list. It is absolutely contraindicated in Stokes-Adams syndrome.

There are however some cases, especially those with transudations, when digitalis may be carefully tried even though high tension be present. It is sometimes of advantage to combine digitalis with the nitrites although they are said to be physiologically incompatible.

Still another drug that is of great value in conditions such as have been described is diuretin. This may be given in capsule or

tablets, grs. x. three times daily. There is only one caution to express in the use of this drug. It does not act well when the kidneys are the seat of chronic inflammatory changes; in fact, actual harm may be done by administering the drug under such conditions.

For the pain in aneurysm nothing (except, of course, morphine) is so valuable as iodide of potassium. Patients who are suffering agony, when put to bed and given KI grs. x. three times a day, soon lose all the distressing symptoms. This applies particularly to aneurysms of the arch of the aorta.

When the sclerosis has affected the cerebral arteries to such an extent that symptoms result, the case is, as a rule, exceedingly grave. Not much can be done except to relieve the headaches and keep down the blood pressure, if this is high, by means of rest in bed, the iodides, aconite, or the nitrites. The cases of transient monoplegias or hemiplegias can be much relieved by careful hygienic measures and judicious administration of drugs. Much ingenuity is sometimes required to overcome the idiosyncracies of

patients, but care and patience will succeed in surmounting all such difficulties.

The treatment of intermittent claudication is the treatment of arteriosclerosis in general. Sometimes the circulation in the affected leg or legs is much helped by daily warm foot baths. Light massage might be tried and the galvanic current may be used once or twice daily.

There are a few distressing symptoms that occur usually late in the disease, when complications have already occurred, which frequently baffle the therapeutic skill of the physician. The chief of these, insomnia, dyspnoea, and headache may not be late manifestations, but insomnia and headache are frequently associated with the moderately advanced stages of arteriosclerosis. At times all the symptoms seem to be due to the high tension the relief of which causes them to disappear. There are unfortunately times when high tension is not responsible for the headache and insomnia. Under these circumstances, such drugs as trional, veronal, amylene hydrate, ammonol, etc., may be tried until one is

found to give sleep. For the headaches phenacetin alone or in combination with caffein and bromide of sodium may be tried. Acetanilid, cautiously used, is at times of value. There have been cases of arteriosclerosis with low blood pressure accompanied by severe headaches that have been relieved by ergot. Codein should be used with care and morphine only as a very last resource.

Great care must always be exercised in giving drugs that depress the circulation for it is easily conceivable that more harm than good can come from injudicious drugging.*

* Quite recently sodium sulphocyanate is said by some to give excellent results in the treatment of arteriosclerosis. It is given in doses of a fraction of a grain to one grain, well diluted with water, three times daily. Those who recommend the drug emphasize the necessity of care in the administration. It should not be given for any length of time or in large doses. Frequent blood pressure estimations should also be made, and the patient should be carefully watched. The drug is a poison. Experimentally ***in vitro*** it has the power, even in very dilute solution, of dissolving the salts of calcium.

CHAPTER XI.

PRACTICAL SUGGESTIONS.

The time spent in obtaining a careful history of a case is time well spent. Often the diagnosis can be made from the history alone, the physical examination merely adding confirmation to the data already obtained.

The younger the patient who has arteriosclerosis, the more probable is it that syphilis is the etiological factor. A denial of infection should have little weight if the history of possible exposure is present. Miscarriages in a woman should arouse the suspicion of lues in her husband.

There are various ways of examining a patient but there is only one right way; the examination should be made on the bare skin. However skillful one may be in the art of physical diagnosis, he can gather few accurate data by examining over the clothes even if he use a phonendoscope.

The immoderate eater is laying up for himself a wealth of trouble at the time when he

can least afford to bear it. The ounce of advice in time is worth more to him than the pounds of medicine later.

It is a wise maxim never to drive a horse too far. Apply that to the human being and the rule holds equally well.

There may be no symptoms in a case of advanced arteriosclerosis. Do not on that account neglect to advise a patient in whom the disease is accidentally discovered.

Many a man owes a debt of gratitude to the life insurance examiner. He rarely feels grateful.

When a competent ophthalmologist refers a case to a general practitioner with the statement that he believes from the appearance of the fundus of the eye that arteriosclerotic changes are present over the body, the case should be most carefully examined. The earliest diagnoses are not infrequently made by the ophthalmologist.

It is the part of wisdom never to have such a firmly preconceived idea of the diagnosis that facts observed are perverted in order to fit into the diagnosis. Let the facts speak for themselves.

Beware of the snap diagnosis. Even in a case of well marked arteriosclerosis when the diagnosis seems to be written in large letters all over the patient, go through the routine. Nine times out of ten this may seem needless. The tenth time it saves your conscience and reputation. Always consider that you are examining a tenth case.

Gradual loss of weight in a person over fifty years old should arouse the suspicion of arteriosclerosis.

Do not call the nervous symptoms displayed by a middle-aged man or woman neurasthenia until you have ruled out all organic causes, particularly arteriosclerosis.

When palpating the radial artery, always use both hands according to the method already described. Pay attention to the superficial or deep situation of the artery.

The examination of one specimen of urine does not give much information especially if it should be found to contain no abnormal elements. Fairly accurate data may be gathered from the mixed night and morning urine; most accurate data from the twenty-

four hour specimen. To be of any real value there should be frequent examinations of the day's excretion.

In measuring the day's output a good rule is as follows: Begin to collect urine after the first morning's micturition and collect all including the first quantity passed the next morning. It is best to examine the centrifugated urine for casts even though no albumen be present. It is useless to look for casts in an alkaline urine.

Casts are not infrequently found in chemically normal urine from a middle-aged patient.

Blood pressure readings should always be taken with the patient in the same posture at every estimation. At the first examination it is advisable to take readings from both brachial arteries. Let the patient sit comfortably and relax all muscles.

As a rule there is no anomaly of the urinary secretion, yet one must constantly note the amount passed in twenty-four hours and the frequency of micturition.

Differentiate as soon as possible between the uncompensated heart caused by valvular disease and that caused by arteriosclerosis.

There is a difference in prognosis. Both give the same symptoms, and are treated similarly until compensation returns; thereafter the management of the two forms is different.

Aortic incompetence that comes on late in life is generally the result of curling of the free margins of the valves caused by arteriosclerosis. Prognosis is grave because of the fact that the heart muscle also is the seat of degenerative changes and compensatory hypertrophy is established with difficulty.

When laying down a regime for a patient, consider his disposition, and individualize the treatment. Remember that exercise is an essential feature of the hygiene of the patient's life but do not forget to be explicit about the amount and character of the permissible exercise.

In the prophylaxis of arteriosclerosis, a rational mode of living is the all-important factor. As a rule, the less meat one eats, the less is the liability of arterial degeneration as age advances. The exceptions to this rule are many, and probably depend

upon the character of the "vital rubber" with which the individual begins life.

The diet in well marked cases of arteriosclerosis should be carefully selected with regard to its nutritive and non-irritating character. Animal proteids should be sparingly used. Milk should have an important place in the dietary.

No drug relieves the pain of uncomplicated aneurysm as surely as iodide of potassium.

Iodides frequently upset the stomach. Be cautious in the use of them. The irritable stomach may turn the scales against your patient.

Use cardiac stimulants with care and judgment. If all the valuable ammunition is used up at first, the fight will be lost.

When you want to use digitalis, remember two important points. (1) The arteriosclerotic heart is one scarred with patches of fibrous myocarditis, and hence is no longer a heart that can respond with every fibre. (2) Digitalis contracts the arterioles and thus increases the peripheral resistance.

Remember that in the uncompensated heart morphine not only eases the oppressive

dyspnoea, but also steadies and stimulates the heart.

See to it that the patient has a daily movementof the bowels. In the early stage try the effect of the mineral waters such as Pluto, or Hunyadi Janos, or artificial Carlsbad salts (Sprudel salts). These last can be made as follows: Sodium chloride, ʒi, sodium bicarbonate, ʒii; sodium sulphate, ʒiv. Take two tablespoonsful of this in a glass of hot water before breakfast. Should these not succeed, assist the action of the drugs by the use of enemata. The pill of aloin, strychnine sulphate, and extract of cascara, with the addition of a small quantity of hyoscyamus, is a mild tonic purgative. In cases of constipation with high tension, there is no drug as valuable as calomel or one of the other mercurials.

Never give Epsom salts unless copious watery stools are desired to deplete effusion into the serous cavities or into the subcutaneous tissue.

Chronic constipation increases the gravity of the prognosis.

In case of suppression of urine and anasarca, hot air packs are of value. The patient

may be wrapped in a hot wet sheet and covered with blankets. I do not believe in administering pilocarpine to assist the sweating.

Remember to treat the patient and not the disease. The careful hygienic and dietetic treatment, combined with the least amount of drugging, is the best and most rational method of treatment.

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